enclosed room. . . . Measurements were correlated with attitudes toward smoke exposure.

For the entire group of nonsmokers, there were no significant changes in heart rate or diastolic blood pressure during the smoke exposure, but systolic blood pressure was significantly increased at 5 minutes of exposure; at 20 minutes of exposure it had returned to preexposure values.

When results were examined according to nonsmokers attitudes, those who "disliked" being exposed had significantly greater heart rates than those who were "indifferent" at all measurements; blood pressures between the two groups did not differ significantly.

Samet, J.M. (1988)[32]

> While these effects [relating to oxygen transport, heart and blood pressure increases, etc.] of carbon monoxide and nicotine may impair performance, exposures to environmental tobacco smoke are generally at concentrations below which physiological effects would be expected. (p. 12, col. 1)

Shephard, R.J., Collins, R. and Silverman, F. (1979)[33]

The responses of healthy men and women were measured during exercise performance while exposed to PTS vs. a sham exposure. Among several other variables, heart rate was measured.

During a preexposure period, those subjects who were to undergo PTS exposure had higher heart rates than the sham subjects. However, actual exposure was associated with a smaller heart rate increase compared to the sham condition.

> The heart rate was higher before the experimental than before the sham exposures. . . . However, while actually exposed to the cigarette smoke both the increment of heart rate and the absolute heart rate were less than in the corresponding sham exposure. (p. 285)

The authors suggested that these heart rate changes might be related to a subjective anxiety or hyperventilation reaction, rather than an actual physiological response to PTS.

<u>Weber-Tschopp, A., Fischer, T. and Grandjean, E. (1976)</u>[34]

> Thirtythree subjects were exposed in a climatic chamber to cigarette smoke (side stream) produced by a smoking machine. . . . $\%FEV_1/VC$, MMF and heart rate were not significantly affected during exposure. (p. 277)

<u>Weber, A., Fischer, T. and Grandjean, E. (1979)</u>[35]

> In a first study subjects were exposed for 1 hr to constant cigarette smoke concentrations corresponding to 5 or 10 ppm CO. Annoyance, subjective eye irritations, and eye blink rate increase in both conditions during the first 30 min of exposure. Respiratory frequency and heart rate variability are not altered. (p. 205)

Winneke, G., Plischke, K., Roscovanu, A. and Schlipkoeter, H.-W. (1984)[36]

> Neither blood pressure-values, nor heart-rates or fingerpulse-volume were influenced by exposure to tobacco-smoke. This correspond to the fact that nicotine-intake from passive smoking is negligible. Depth and rate of breathing were not altered either. (p. 353)
>
> As for carbon monoxide (CO) there was pronounced uptake in terms of COHb for the high exposure-condition only. In absolut terms, however, the measured values correspond to those found in non-smoking urban populations, and are well below levels considered critical for persons with cardio-vascular impairment. (p. 354)

Winneke, G., Neuf, M., Roscovanu, A. and Schlipköter, H.-W. (1990)[37]

Physiological measurements were obtained from nonsmokers, while they were in an exposure chamber in which another individual was smoking. No significant effects were reported for heart rate or blood pressure. The authors reported that "cardiorespiratory variables were not affected by ETS exposure." (p. 173)

> In taking CO as the basis for comparison our cardiovascular findings are consistent with those of others, who, at even higher levels of ETS-exposure did not observe exposure-related increase of either heart-rate or blood-pressure. Nicotine-intake at such ETS-levels is likely to be too low for cardiovascular changes to be expected. (p. 181)

PTS or CO exposure -- angina patients and other compromised individuals

Claims are sometimes made that PTS, or specifically CO in PTS, aggravates cardiovascular disease in people with pre-existing illness. Such claims typically stem from a limited and largely discredited report by Wilbert Aronow that angina patients experienced chest pain sooner when exercising in the presence of PTS.[38] However, a closer examination of the literature reveals a body of data that raises questions about whether PTS, or CO at levels reported to be in PTS, has significant adverse cardiovascular effects.

Hinderliter, A.L., Adams, K.F., Price, C.J., Herbst, M.C., Koch, G. and Sheps, D.S. (1989)[39]

> In conclusion, low-level CO exposure is not arrhythmogenic in patients with coronary heart disease and no ventricular ectopy at baseline. (p. 89)

McNicol, M.W. and Turner, McM. (1983)[40]

> There was no change in the mean oxygen uptake at the onset of angina with any intervention. . . . These results are in contrast with previous reports of the effects of smoking and carbon monoxide on exercise performance in angina pectoris. However in all of these other studies a subjective rather than objective end point was used. We suggest that an objective assessment is essential and that oxygen uptake at the onset of angina is useful and relatively easy to measure.

Shephard, R.J., Collins, R. and Silverman, F. (1979)[41]

This study was an attempt to determine if asthmatic subjects have an increased sensitivity to PTS exposure. Respiratory symptoms, pulmonary function and heart rates were measured. Although some slight effects (e.g., a small heart rate increase) were reported, these were considered to be of "doubtful biological importance" and most likely due to an emotional, rather than directly physiological, response to the PTS exposure.

> There was also some evidence of arousal and/or emotional excitement, including a slight tachycardia (at 80-min exposure, $P<0.05$) and a slight increase of forced vital capacity ($P<0.05$ at 90-min exposure). However, dynamic lung volumes . . . were unaltered. . . . Our data thus do not suggest that asthmatic subjects have an unusual sensitivity to cigarette smoke. (p. 392)
>
> The physiological changes observed in normal subjects during smoke exposure, although occasionally reaching conventional levels of statistical significance, were of doubtful biological importance (Pimm et al., 1978; Shephard et al., 1979a). Findings included some increase of heart rate and respiratory minute volume, probably of emotional origin, a tendency of increase in functional residual capacity and residual volume in some experiments, and small decreases of dynamic lung volumes. The asthmatic subjects also showed emotional reactions to the cigarette smoke, including the tachycardia, and possibly the preexposure increase of FRC and TLC. (pp. 399, 401)
>
> We would thus conclude that the specific sensitivity of asthmatic subjects is not a major consideration when determining air quality criteria for rooms contaminated by cigarette smoke. (p. 402)

Sheps, D.S., Adams, K.F., Bromberg, P.A., Goldstein, G.M., O'Neil, J.J., Horstman, D. and Koch, G. (1987)[42]

> In conclusion, there is no clinically significant effect of 3.8% COHb (representing a 2.2% increase from resting values) on the cardiovascular system in this study. (p. 108)

REFERENCES

1. Dobson, A.J., Alexander, H.M., Heller, R.F. and Lloyd, D.M., "Passive Smoking and the Risk of Heart Attack or Coronary Death," The Medical Journal of Australia 154: 793-797, 1991

2. Helsing, K.J., Sandler, D.P., Comstock, G.W. and Chee, E., "Heart Disease Mortality in Nonsmokers Living With Smokers," American Journal of Epidemiology 127(5): 915-922, 1988.

3. Hirayama, T., "Lung Cancer in Japan: Effects of Nutrition and Passive Smoking." In: Lung Cancer: Causes and Prevention. M. Mizell and P. Correa (eds.). New York, Verlag Chemie International, Chapter 14, 175-195, 1984.

4. Lee, P.N., Chamberlain, J. and Alderson, M.R., "Relationship of Passive Smoking to Risk of Lung Cancer and Other Smoking-Associated Diseases," British Journal of Cancer 54: 97-105, 1986.

5. Svendsen, K.H., Kuller, L.H., Martin, M.J. and Ockene, J.K., "Effects of Passive Smoking in the Multiple Risk Factor Intervention Trial," American Journal of Epidemiology 126(5): 783-795, 1987.

6. Wells, A.J., "An Estimate of Adult Mortality in the United States from Passive Smoking," Environment International 14(3): 249-265, 1988.

7. Kawachi, I., Pearce, N.E. and Jackson, R.T., "Deaths from Lung Cancer and Ischaemic Heart Disease Due to Passive Smoking in New Zealand," New Zealand Medical Journal 102(871): 337-340, 1989.

8. Kristensen, T.S., "Cardiovascular Diseases and the Work Environment. A Critical Review of the Epidemiologic Literature on Chemical Factors," Scand J Work Environ Health 15: 245-264, 1989.

9. Glantz, S.A. and Parmley, W.W., "Passive Smoking and Heart Disease: Epidemiology, Physiology, and Biochemistry," Circulation 83(1): 1-12, 1991.

10. Steenland, K., "Passive Smoking and the Risk of Heart Disease," JAMA 267(1): 94-99, 1992.

11. U.S. Department of Health and Human Services, The Health Consequences of Involuntary Smoking: A Report of the Surgeon General. Publication No. DHHS (CDC) 87-8398, Washington, D.C.,

U.S. Government Printing Office, 1986.

12. Committee on Passive Smoking, Board on Environmental Studies and Toxicology, National Research Council, National Academy of Sciences, Environmental Tobacco Smoke: Measuring Exposures and Assessing Health Effects. Washington, D.C., National Academy Press, 1986.

13. Wexler, L.M., "Environmental Tobacco Smoke and Cardiovascular Disease: A Critique of the Epidemiological Literature and Recommendations for Future Research." In: Environmental Tobacco Smoke: Proceedings of the International Symposium at McGill University 1989. D.J. Ecobichon and J.M. Wu (eds.). Lexington, Mass., Lexington Books, D.C. Heath and Company, Chapter 8, 139-152, 1990.

14. Weetman, D.F. and Munby, J., "Environmental Tobacco Smoke (ETS) and Cardiovascular Disease." In: Indoor Air Quality and Ventilation. F. Lunau and G.L. Reynolds (eds.). London, Selper Ltd., 211-216, 1990.

15. Thiery, J. and Cremer, P., "Coronary Heart Disease and Involuntary Smoking," Paper presented at: Toxicology Forum (Session on "Environmental Tobacco Smoke: Science and Meta-Science"), Budapest, Hungary, June 19, 1990.

16. Armitage, A.K., "Environmental Tobacco Smoke and Coronary Heart Disease." In: Other People's Tobacco Smoke. A.K. Armitage (ed.). Beverly, E. Yorks, U.K., Galen Press, Chapter 7, 109-116, 1991.

17. Lee, P.N., "Weaknesses in Recent Risk Assessments of Environmental Tobacco Smoke," Environmental Technology 12(3): 193-208, 1991.

18. Adlkofer, F.X., Scherer, G., Von Meyerinck, L., Von Maltzan, Ch. and Jarczyk, L., "Exposure to ETS and its Biological Effects: A Review." In: Present and Future of Indoor Air Quality: Proceedings of the Brussels Conference, 14-16 February 1989. C.J. Bieva, Y. Courtois and M. Govaerts (eds.). New York, Elsevier Science Publishing Company, Inc., 183-196, 1989.

19. Cole, P., "Smoking Habits and Carbon Monoxide." In: Smoking and Arterial Disease, R.M. Greenhalgh (ed.). Pittman Medical, Chapter 10, 74-83, 1981.

20. Gori, G.B. and Mantel, N., "Mainstream and Environmental Tobacco Smoke," Regulatory Toxicology and Pharmacology 14: 88-105, 1991.

21. Malmfors, T., Thorburn, D. and Westlin, A., "Air Quality in Passenger Cabins of DC-9 and MD-80 Aircraft," Environmental Technology Letters 10: 613-628, 1989.

22. Proctor, C.J., Warren, N.D., Bevan, M.A.J. and Baker-Rogers, J., "A Comparison of Methods of Assessing Exposure to Environmental Tobacco Smoke in Non-Smoking British Women," Environment International 17: 287-297, 1991.

23. Rylander, R., "Workshop Perspectives." In: ETS--Environmental Tobacco Smoke. Report from a Workshop on Effects and Exposure Levels, R. Rylander, Y. Peterson and M.-C. Snella (eds.). Chapter 5, 143-145, University of Geneva, Switzerland, March 15-17, 1983.

24. Schievelbein, H. and Richter, F., "The Influence of Passive Smoking on the Cardiovascular System," Preventive Medicine 13: 626-644, 1984.

25. Committee on Passive Smoking, Board on Environmental Studies and Toxicology, National Research Council, National Academy of Sciences, Environmental Tobacco Smoke: Measuring Exposures and Assessing Health Effects. Washington, D.C., National Academy Press, 1986.

26. Fischer, T., Weber, A. and Grandjean, E., "Extent and Effect of Air Pollution by Tobacco Smoke Under Experimental Conditions and in Restaurants," (translated from German), Schweiz Blatt Luft 45: 3-8, 1978.

27. Harke, H.-P. and Bleichert, A., "On the Problem of Passive Smoking," Int Arch Arbeitsmed 29: 312-322, 1972.

28. Hulka, B.S., "The Health Consequences of Environmental Tobacco Smoke," Environmental Technology Letters 9: 531-538, 1988.

29. International Collaborative Group, "International Collaborative Study on Juvenile Hypertension. 2. First Follow-Up Report," Bulletin of the World Health Organization 62(1): 121-132, 1984.

30. Pimm, P.E., R.J. Shephard and F. Silverman, "Physiological Effects of Acute Passive Exposure to Cigarette Smoke," Archives of Environmental Health 33(4): 201-213, 1978.

31. Rummel, R.M., Crawford, M. and Bruce, P., "The Physiological Effects of Inhaling Exhaled Cigarette Smoke in Relation to Attitude of the Nonsmoker," The Journal of School Health 45(9): 524-529, 1975.

32. Samet, J.M., "Involuntary Exposure to Tobacco Smoke," Annals of Sports Medicine 4(1): 1-15, 1988.

33. Shephard, R.J., Collins, R. and Silverman, F., "Responses of Exercising Subjects to Acute 'Passive' Cigarette Smoke Exposure," Environmental Research 19: 279-291, 1979.

34. Weber-Tschopp, A., Fischer, T. and Grandjean, E., "Physiological and Psychological Effects of Passive Smoking," (translated from German), Int. Arch. Occup. Environ. Hlth. 37(4): 277-288, 1976.

35. Weber, A., Fischer, T. and Grandjean, E., "Passive Smoking in Experimental and Field Conditions," Environmental Research 20(1): 205-216, 1979.

36. Winneke, G., Plischke, K., Roscovanu, A. and Schlipkoeter, H.-W., "Patterns and Determinants of Reaction to Tobacco Smoke in an Experimental Exposure Setting." In: Indoor Air: Radon, Passive Smoking, Particulates and Housing Epidemiology. B. Berglund, T. Lindvall and J. Sundell (eds.). Vol. 2, 351-356, 1984.

37. Winneke, G., Neuf, M., Roscovanu, A. and Schlipköter, H.-W., "Psychophysiological Response to Environmental Tobacco Smoke in an Experimental Social Setting." In: Indoor Air Quality. H. Kasuga (ed.). Berlin, Springer-Verlag, 173-183, 1990.

38. Aronow, W.S., "Effect of Passive Smoking on Angina Pectoris," New England Journal of Medicine 299(1): 21-24, 1978.

39. Hinderliter, A.L., Adams, K.F., Price, C.J., Herbst, M.C., Koch, G. and Sheps, D.S., "Effects of Low-Level Carbon Monoxide Exposure on Resting and Exercise-Induced Ventricular Arrhythmias in Patients with Coronary Artery Disease and No Baseline Ectopy," Archives of Environmental Health 44(2): 89-93, 1989.

40. McNicol, M.W. and Turner, McM., "Oxygen Uptake at the Onset of Angina Pectoris: Effects of Nicotine and Carbon Monoxide," Clinical Science 65(3): 248, 1983.

41. Shephard, R.J., Collins, R. and Silverman, F., "'Passive' Exposure of Asthmatic Subjects to Cigarette Smoke," Environmental Research 20(2): 392-402, 1979.

42. Sheps, D.S., Adams, K.F., Bromberg, P.A., Goldstein, G.M., O'Neil, J.J., Horstman, D. and Koch, G., "Lack of Effect of Low Levels of Carboxyhemoglobin on Cardiovascular Function in Patients with Ischemic Heart Disease," Archives of

Environmental Health 42(2): 108-116, 1987.

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Response to Question 2a(iii) (continued):

Adult Respiratory Disease and Symptoms

The majority of the studies on spousal smoking and respiratory disease other than cancer in nonsmoking adults have been conducted in the home. Only a few studies have examined the possible relationship between reported exposures to PTS in the workplace and the respiratory health of nonsmoking adults.[1-6] The studies that have been conducted on this issue have reported contradictory results. There are also problems with the methodology utilized in these studies. For example, one often-cited study reported an association between workplace exposure to PTS and small-airways dysfunction in adults,[1] but the authors have been criticized heavily for questionable data acquisition and analysis.[7-15]

Other researchers who have reviewed the reported findings concerning the possible relationship between PTS and respiratory symptoms and diseases in adults also contend that the results are mixed and inconclusive. For example, French researchers, Laurent, et al., have commented that purported long-term health effects from exposure to PTS are difficult to demonstrate in healthy adults and that the results of the epidemiological studies are "sometimes conflicting and often open to question."[16] One investigator has conceded that "the effect of passive smoking on respiratory

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infections in adults has not been well characterized and reports of its effects on chronic respiratory disease in adults have been inconsistent."[17]

In his summary of the studies of respiratory symptoms and disease in adults through early 1990, American investigator Dr. Philip Witorsch noted that "4 of 8 [reported] an increased frequency, 4 of 8 no increased frequency." He stated that, in addition to "all of the problems" with the studies, "these results are too variable to permit any conclusion of association."[18] As his summary demonstrates, the conclusion of the participants at the 1983 U.S. National Institutes of Health workshop on PTS exposure, namely, that the possible effect from PTS "varies from negligible to quite small," is still pertinent.[19]

In addition to inconsistent and variable conclusions, confounding factors are not adequately controlled for in these studies. The same confounders that reportedly affect studies in the home may also play a role in workplace exposure studies. Although some of these confounders, such as gas stove usage, may not directly apply to the indoor air quality of the workplace, exposures in the home to such sources may influence the respiratory health status of the populations studied in the workplace. Potential confounders for adult respiratory health include gas stove usage in the home,[19-20] outdoor air pollution,[21-22]

socioeconomic status,[23,19] damp housing[19,24-25] and exposure to various indoor air contaminants.[19]

A Discussion of the Published Studies

In 1980, White and Froeb evaluated the possible effect of long-term "passive" smoking and long-term voluntary smoking on specific indexes of pulmonary function in 2,100 middle-aged adults.[1] Eighty-three percent of the subjects held professional, managerial, or technical positions, and the remainder were blue-collar workers. Subjective reports of exposure to active and "passive" smoking were obtained by a self-administered questionnaire.

Nonsmoking subjects indicated on the questionnaire whether smoking was permitted in their working environments. In addition, a portable carbon monoxide analyzer was placed on top of the desk or in the working area of 40 randomly selected nonsmokers who had indicated that they worked in an environment without smoke and 40 similarly selected nonsmokers who had indicated that the air in their working area contained smoke from co-workers. The authors reported that "compared with nonsmokers who worked in environments where there was no smoking, both the men and the women in the other five groups had significantly lower values for FEF 25 to 75 per cent and FEF 75 to 85 per cent." Furthermore, the authors suggested that "the passive smokers not only scored significantly lower than

their nonsmoking counterparts but also fell into the same state of impaired performance as the noninhalers and light smokers."

The White and Froeb study has been criticized for numerous reasons.[7-10] For example, a physician at a U.S. medical school questioned their use of carbon monoxide as an index of smoke exposure, contending that they "do not have reliable estimates of the smoke exposure in the environment of their nonsmokers" because carbon monoxide is not unique to tobacco smoke.[10] A British reviewer shared the physician's view that their findings "relate to an index which is contentious and certainly not an accepted reliable indicator of an increased health risk."[11] In addition, White and Froeb themselves noted that the average values of the pulmonary tests of nonsmokers exposed to tobacco smoke "were not notably different" from the values suggested as normal by a specialist in this area.[12]

Perhaps the most telling criticisms of the study were voiced by Dr. Michael Lebowitz of the University of Arizona at an annual joint meeting of the American Lung Association/American Thoracic Society and in a subsequent letter published in the U.S. Congressional Record.[13-14] During a forum at the ALA meeting, Dr. Lebowitz stated that he had concluded, from his own extensive review of the study and from an interview with White, that the study was "improperly designed" from an epidemiological point of

view. He noted that there were problems "inherent" in the study, including the selection of the study group and the measurement of smoke in the workplace. Dr. Lebowitz also expressed concern that the statistical significance of the data appeared to depend on the unexplained omission of data for 3,000 people who were originally included in the study. Based upon these considerations, Dr. Lebowitz urged that the study not be used to support the claim that PTS affects the lung function of adults in the workplace. Dr. Lebowitz again took issue with the White and Froeb study in 1984 in a paper he presented at the Vienna Symposium on Passive Smoking. He contended:

> Even with a biased population, poor study design, and incorrect statistical evaluation, there were no clear-cut, consistent, medically meaningful differences between passive smokers and groups of nonsmokers; a corrected statistical analysis strengthened this conclusion.[15]

In 1984, Kentner, et al., published results of a study assessing the possible influence of reported "passive" smoking on pulmonary function in 1,351 men and women.[2] All subjects were white-collar workers, and the majority were employed in sedentary office jobs. Each subject filled out a self-administered questionnaire, and pulmonary function tests were administered to all of the subjects.

The authors reported that for one pulmonary function parameter, FVC, the values were similar in all five subgroups of male subjects. Only the current smoking group differed significantly from the three groups of nonsmokers among females. Similar results were reported for $FEF_{25/75}$ and for FEV_1. $FEF_{75/85}$ values reportedly differed significantly in male ex-smokers and current smokers as compared with non-smokers and men reportedly exposed to PTS. For $MEF_{25/75}$, male ex-smokers differed from both male "passive" and current smokers, while female non-smokers and ex-smokers differed from current smokers. Reported "passive" smokers showed essentially no decrease in lung function parameters. The authors concluded that "it seems that the passive inhalation of tobacco smoke at home or in the workplace by healthy individuals probably does not lead to any essential impairment of pulmonary function." The authors also reported these findings in 1988 and 1990 publications.[3-4]

In 1989, Kentner, et al., reported on their investigations of pulmonary function in adults reportedly exposed to "passive" smoking in the workplace.[5] In the years 1983-1984, they examined 1,364 employees with healthy lungs from three different companies. Information concerning tobacco smoke exposure was taken from a standardized questionnaire.

Bronchopulmonary function was checked with the flow-volume curve. The authors again reported that "everyday passive exposure to smoke in the office or at home does not cause a significant impairment of lung function in adults with healthy lungs." The authors also raised the question as to the extent to which the reported "passive" smoking-induced impairment by White and Froeb and others "is at all relevant pathophysiologically." They wrote that "it may be assumed that, under normal, everyday conditions, exposure of adults with healthy lungs to passive smoke does not lead to relevant pathological changes in the bronchopulmonary function in the sense of adverse effects."

In a 1991 study, White, Froeb and Kulik monitored CO levels (as an index of cigarette smoke in the workplace) and analyzed diary entries on respiratory symptoms, eye irritation, chest colds, and lost days from work due to respiratory illness in 40 nonsmokers reportedly chronically exposed to PTS in the workplace.[6]

Exposure to environmental pollution, smoking and physical characteristics were assessed from a self-administered questionnaire completed during the course of the study. The self-reported PTS exposed subjects met the same characteristics as the nonexposed nonsmokers, only they had reported exposures to PTS in the workplace for at least one year. To obtain "objective measures

of tobacco smoke concentrations," a single trained technician visited the work site on three occasions. The CO levels were recorded continuously for 24 hours. Results from the three days were then averaged.

Each subject responded to a questionnaire developed by the American Thoracic Society during one of the three visits. The questionnaire solicited information on respiratory symptoms including chronic cough, chronic phlegm, breathlessness, chest colds and chest illnesses, number of days missed from work due to chest cold or chest illness, and eye irritation.

The authors concluded that "chronic exposure to tobacco smoke in the work place produces significant eye irritation and increases respiratory symptoms and the incidence of chest colds." However, the authors failed to control for various confounding factors that might have affected this reported association. For example, dietary factors, gas cooking at home, and bioaerosols in the workplace were not controlled for in this study. Furthermore, carbon monoxide is not an accurate assessment of exposure to PTS because tobacco smoke is not the only potential source of CO in the indoor environment.[10] Therefore, the authors' conclusion that PTS in the workplace has deleterious effects on the respiratory health of adults is not justified by the scientific data.

Conclusion

The studies on workplace exposure to PTS and the respiratory health of nonsmoking adults are inconsistent in their reported results. Furthermore, the single study which reports an association between workplace exposure to PTS and small airways dysfunction in adults has been heavily criticized for questionable data acquisition and analysis. Thus, the data do not support the conclusion that PTS exposure in the workplace contributes to chronic respiratory disease and symptoms in nonsmoking adults.

REFERENCES

1. White, J.R. and Froeb, H.F., "Small-Airways Dysfunction in Nonsmokers Chronically Exposed to Tobacco Smoke," The New England Journal of Medicine 302 (13): 720-723, 1980.

2. Kentner, M., Triebig, G. and Weltle, D., "The Influence of Passive Smoking on Pulmonary Function - A Study of 1,351 Office Workers, Preventive Medicine 13(6): 656-669, 1984.

3. Kentner, M. and Weltle, D., "Passive Tobacco Smoke Inhalation and Lung Function in Adults." In: Indoor and Ambient Air Quality. R. Perry and P. Kirk (eds.). London, Selper Ltd., 232-241, 1988.

4. Kentner, M. and Weltle, D., "Are There Any Impairments of Maximal Expiratory Flow-Volume Curves by Passive Smoking?" In: Indoor Air Quality. H. Kasuga (ed.). Berlin, Heidelberg, Springer-Verlag, 153-166, 1990.

5. Kentner, M., Weltle, D. and Valentin, H., "Passive Smoking and Lung Function in Adults," Arbeitsmedicine Sozialmedicine Praventivmedicine (24): 8-13, 1989.

6. White, J.R., Froeb, H.F. and Kulik, J.A., "Respiratory Illness in Nonsmokers Chronically Exposed to Tobacco Smoke in the Work Place," Chest 100(1): 39-43, 1991.

7. Adlkofer, F., Scherer, G. and Weimann, H., "Small-Airways Dysfunction in Passive Smokers," New England Journal of Medicine 303(7): 392, 1980.

8. Huber, G., "Small-Airways Dysfunction in Passive Smokers," New England Journal of Medicine 303(7): 392, 1980.

9. Freedman, A., "Small-Airways Dysfunction in Passive Smokers," New England Journal of Medicine 303(7): 393, 1980.

10. Aviado, D., "Small-Airways Dysfunction in Passive Smokers," New England Journal of Medicine 303(7): 393, 1980.

11. Lee, P.N., "Passive Smoking," Food Chemistry and Toxicology 20: 223-229, 1982.

12. White, J. and Froeb, H., "Small-Airways Dysfunction in Passive Smokers," New England Journal of Medicine 303(7): 393-394, 1980.

13. Lebowitz, M., "Forum: Second Hand Smoke -- How Dangerous?," Presentation to the American Lung Association/American Thoracic Society Annual Meeting, Los Angeles, California, May 17, 1982.

14. Fountain, L.H., Statement Re: "White-Froeb Study Discredited by Scientists." In: U.S. Congressional Record, 97th Congress, 2nd Session, E5252-5254, December 16, 1982.

15. Lebowitz, M., "Influence of Passive Smoking on Pulmonary Function: A Survey," Preventive Medicine 13(6): 645-655, 1984.

16. Laurent, A.M., Courtois, Y.A., Pretet, S. and Claude, J.R., "Evaluation of Exposure to Ambient Tobacco Smoke: A Few Concrete Examples Concerning the Value of Biological Atmospheric Indicators." In: Present and Future of Indoor Air Quality. C.J. Bieva, Y. Courtois, M. Govaerts (eds.). Amsterdam, Excerpta Medica, 103-110, 1989.

17. Graham, N.M.H., "The Epidemiology of Acute Respiratory Infections in Children and Adults: A Global Perspective," Epidemiologic Reviews 12: 149-178, 1990.

18. Witorsch, P., "Health Effects of Environmental Tobacco Smoke." In: Toxicology Forum: 1990 Annual Winter Meeting. Washington D.C., 212, February 19-21, 1990.

19. U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, Report of Workshop on Respiratory Effects of Involuntary Smoke Exposure: Epidemiologic Studies, May 1-3, 1983, December, 1983.

20. Koo, L.C. and Ho, J.H.C., "Sources of Nitrogen Dioxide in Homes and Its Association with Respiratory Illness in Hong Kong." In: Indoor Air Quality and Ventilation. London, Selper Ltd., 287-294, 1990.

21. Detels, R., Tashkin, D.P., Sayre, J.W., Rokaw, S.N., Massey, F.J., Coulson, A.H. and Wegman, D.H., "The UCLA Population Studies of CORD: X. A Cohort Study of Changes in Respiratory Function Associated with Chronic Exposure to SOx, NOx, and Hydrocarbons," American Journal of Public Health 81(3): 350-359, 1991.

22. Xu, X., Dockery, D.W. and Wang, L., "Effects of Air Pollution on Adult Pulmonary Function," Archives of Environmental Health 46(4): 198-206, 1991.

23. Holma, B. and Winding, O., "Housing, Hygiene, and Health: A Study in Old Residential Areas in Copenhagen," Archives of Environmental Health (March/April): 86-93, 1977.

24. Platt, S.D., Martin, C.J., Hunt, S.M. and Lewis, C.W., "Damp Housing, Mould Growth, and Symptomatic Health State," British Medical Journal 298: 1673-1678, 1989.

25. Dales, R.E., Burnett, R. and Zwanenburg, H., "Adverse Health Effects Among Adults Exposed to Home Dampness and Molds," American Review of Respiratory Disease 143(3): 505-509, 1991.

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Response to Question 2a(iii) (continued):

Parental Smoking

While parental smoking studies are not directly related to workplace indoor air quality issues, they comprise a large proportion of the available studies on PTS, and are often cited by those who argue that PTS exposures are associated with respiratory disease and symptoms in nonsmokers. However, the scientific basis for the claim that PTS is associated with respiratory diseases and symptoms in children is difficult to interpret.

A Discussion of the Published Studies

While one study on parental smoking examines respiratory symptoms or illness such as coughs and colds by questionnaire responses from parents,[1] another measures lung function with special equipment at a school or health facility.[2] In the U.S. alone, according to one report, this has led to a situation in which studies of PTS and the respiratory system are "being carried out by at least three different groups, are employing different populations and methodologies and have led to varying conclusions.[3]

Perhaps not surprisingly, studies of parental smoking, each with a different sample size, data collection method, and analysis, tend to yield factually incompatible and contrary

conclusions. For instance, although certain studies have reported adverse findings between parental smoking and respiratory illness in children,[4-35] others have observed no significant relationship.[1,36-51] After a five-year study of over 400 children, for example, a Dutch research group concluded there was "no evidence" that parental smoking had an appreciable effect on respiratory symptoms in school children.[43] A similar conclusion was reached by a group of U.S. researchers, including a critic of smoking, who found "no significant relation" between parental smoking and respiratory symptoms in a study of nearly 400 families with 816 children in three cities.[36]

The contradictory nature of findings on the issue of parental smoking is also apparent in the growing number of studies examining the relationship between parental smoking and children's respiratory or lung function. Although several reports have claimed that parental smoking results in decreased pulmonary function in children,[8,13-14,32,52-60] others have not,[61-64] including those of a U.S. research group who have published a series of studies on this subject.[2,65-67] In 1982, for example, the U.S. group showed that a comparison of body size with lung function eliminated any reported correlation between parental smoking and children's lung function.[2] Two years later, a reanalysis of data from families in their study population again showed that "parental smoking did not have a significant effect on children's pulmonary function; smoking habits

of others in the household (predominantly siblings) did not have any effect either."[66]

Confounding Factors and Other Sources of Bias

The authors of studies reporting adverse effects from PTS exposures among children concede that their conclusions must be viewed with caution because of numerous confounding factors. The potential impact of such factors was given special consideration in the report from a workshop on PTS sponsored by the U.S. National Institutes of Health.[3] After listing numerous such factors, including types of heating used, socio-economic status and demographic and medical characteristies of the study population, the report cautioned "that any study which ignores them will be seriously flawed."

The importance of such factors in evaluating the outcome of research on parental smoking is supported by a number of reports which have shown that the use of gas stoves in the home may be independently associated with respiratory disease[40,65,68-72] and impaired pulmonary performance[73-74] in children. One group of British researchers acknowledged the possible influence of factors such as cross-infection in the home and genetic susceptibility to childhood respiratory illness and symptoms.[11-12] More recently, researchers in Hong Kong reported "a highly significant correlation"

between the frequency of respiratory illnesses of mothers and their children.[75]

Other confounding factors independent of parental smoking have been reported recently in the literature. For example, studies in the United Kingdom have identified damp housing[46,76-79] and paternal occupation[80] as potential explanatory factors for the occurrence of respiratory illness in children. Other recent studies have identified outdoor air pollution,[82-85] infections transmitted during day-care attendance,[49,86-87] decreased breast-feeding[88] and the use of kerosene heaters and woodburning stoves in the home[89-91] as factors related to childhood respiratory disease.

The relevance of dampness in the etiology of respiratory symptoms in children is supported by current research which links dampness with the presence of molds, dust mites, fungi and other allergenic microbes. The growth of fungi and molds in the home is directly related to respiratory symptoms and sensitization reactions in some individuals.[78,92-94] Recent investigations report, moreover, that exposure to PTS does not increase sensitization to common allergens in children.[95-96]

Others have conceded that the reliance of such studies on questionnaires for information about respiratory symptoms casts doubt on their reported findings. In one study that reported a

significant association between parental smoking and respiratory symptoms, for example, it was noted that even "slight changes" in the way the questions were phrased could result "in substantial differences in the type of responses one obtains."[14] Similarly, another study observed that there was a significant difference in the respiratory symptoms reported depending on which parent completed the questionnaire.[24] Authors of another study that reported adverse effects of parental smoking on the respiratory health of children conceded that "since the exposure variables used in these analyses were subject to substantial measurement error, a more refined measurement of personal exposure is required."[29] One researcher who is critical of parental smoking has stated that "quantitative assessment of involuntary exposure of infants and children to ETS has been very imprecise and probably inaccurate."[97]

Studies utilizing seemingly more objective standards such as actual measurements of lung function are also open to criticism. Even reviewers of the literature who are critical of parental smoking concede that the tests used in these studies are "influenced by a large number of variables."[98] They list age, height, and gender of the test subject as well as his or her motivation, cooperation, and effort put forth during the test, the skill and experience of the operator, and the type of instrumentation used as variables that can affect the results of

pulmonary function measurements. The reviewers explain that these problems are especially important in pulmonary function measures taken in children. In 1989, two American co-researchers, Witorsch and Witorsch, reported that "it has been shown that mean pulmonary performance within a single group of children can vary significantly from one spirometry test to the next without any apparent cause" and that it "is noteworthy that such statistically significant differences are similar in magnitude to most of the small decrements in pulmonary function reported in children of smoking parents."[99]

The shortcomings of studies analyzing the relationship between PTS exposure and childhood health were highlighted in a 1988 report by two U.S. investigators who re-examined 30 such studies and evaluated them for their scientific validity.[100] They noted that while several studies of adequate scientific design have reported a statistically significant relationship between PTS exposures and childhood health, "most studies had significant design problems that prevent reliance on their conclusions." The authors concluded that "many questions remain, and future studies should consider important methodological standards to determine more accurately the effect of passive smoking on child health."

Thus, claims that parental smoking plays a causal role in the development of respiratory symptoms and reduced lung function in children are not scientifically justified. Such claims are

typically based upon a single study of a selected symptom (such as cough or wheeze). These kinds of studies invariably fail to consider nutrition, health habits of the family, and other lifestyle variables. Similarly, studies that report reduced lung function in children of smoking parents fail to address the issue of socio-economic status or the potential role of genetic and family traits in pulmonary function capabilities.[64] Moreover, the reductions reported in the literature are small and of uncertain clinical or biological significance, and are contradicted by a number of studies that reportedly have observed no effect of parental smoking on children's lung function.

REFERENCES

1. Camacho, E., Colomer, P.R. and Betoret, J.L.D., "Pulmonary Symptoms and Pulmonary Functional Tests Among Children in Relation to the Area of Residence," European Respiratory Diseases 63(2): 165-166, 1982.

2. Lebowitz, M., Armet, D.B. and Knudson, R., "The Effect of Passive Smoking on Pulmonary Function in Children," Environment International 8: 371-373, 1982.

3. U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health, Report of Workshop on Respiratory Effects of Involuntary Smoke Exposure: Epidemiologic Studies, May 1-3, 1983, December, 1983.

4. Committee on Passive Smoking, Board on Environmental Studies and Toxicology, National Research Council, Environmental Tobacco Smoke: Measuring Exposures and Assessing Health Effects. Washington, D.C., National Academy Press, 1986.

5. U.S. Department of Health and Human Services, Public Health Services, Office on Smoking and Health, The Health Consequences of Involuntary Smoking: A Report of the Surgeon General. DHHS Publication No. (CDC) 87-8398, Washington, D.C., U.S. Government Printing Office, 1986.

6. Bland, M., Bewley, B.R., Pollard, V. and Banks, M.H., "Effect of Children's and Parents' Smoking on Respiratory Symptoms," Archives of Diseases in Childhood 53(2): 100-105, 1978.

7. Bonham, G. and Wilson, R., "Children's Health in Families With Cigarette Smokers," American Journal of Public Health 71(3): 290-293, 1981.

8. Burchfiel, C., Higgins, M.W., Keller, J.B., Howatt, W.F., Butler, W.J. and Higgins, I.T.T., "Passive Smoking in Childhood: Respiratory Conditions and Pulmonary Function in Tecumseh, Michigan," American Review of Respiratory Disease 133(6): 966-973, 1986.

9. Cameron, P. and Robertson, D., "Effect of Home Environment Tobacco Smoke on Family Health," Journal of Applied Psychology 57(2): 142-147, 1973.

10. Chen, Y., "Synergistic Effect of Passive Smoking and Artificial Feeding on Hospitalization for Respiratory Illness in Early Childhood," Chest 95: 1004-1007, 1989.

11. Colley, J., "Respiratory Symptoms in Children and Parental Smoking and Phlegm Production," British Medical Journal II: 201-204, 1974.

12. Colley, J., Holland, W.W. and Corkhill, R.T., "Influence of Passive Smoking and Parental Phlegm on Pneumonia and Bronchitis in Early Childhood," Lancet II: 1031-1034, 1974.

13. Dockery, D., Speizer, F.E., Stram, D.O., Ware, J.H., Spengler, J.D. and Ferris, B.G., "Effects of Inhalable Particles on Respiratory Health of Children," American Review of Respiratory Disease 139: 587-594, 1989.

14. Ekwo, E., Weinberger, M.M., Lachenbruch, P.A. and Huntley, W.H., "Relationship of Parental Smoking and Gas Cooking to Respiratory Disease in Children," Chest 84(6): 662-668, 1983.

15. Fergusson, D., Horwood, L.J., Shannon, F.T. and Taylor, B., "Parental Smoking and Lower Respiratory Illness in the First Three Years of Life," Journal of Epidemiology and Community Health 35(3): 180-184, 1981.

16. Fergusson, D. and Horwood, L.J., "Parental Smoking and Respiratory Illness During Early Childhood: A Six-year Longitudinal Study," Pediatric Pulmonology 1(2): 99-106, 1985.

17. Ferris, B., Dockery, D.W., Ware, J.H., Speizer, F.E. and Spiro, R., "The Six-City Study: Examples of Problems in Analysis of Data," Environmental Health Perspectives 52: 115-123, 1983.

18. Gortmaker, S., Walker, D.K., Jacobs, F.H. and Ruch-Ross, H., "Parental Smoking and the Risk of Childhood Asthma," American Journal of Public Health 72(6): 574-579, 1982.

19. Harlap, S. and Davies, A., "Infant Admissions to Hospital and Maternal Smoking," Lancet I: 529-532, 1974.

20. Kraemer, M., Richardson, M.A., Weiss, N.S., Furukawa, C.T., Shapiro, G.G., Pierson, W.E. and Bierman, C.W., "Risk Factors for Persistent Middle Ear Effusions: Otitis Media, Catarrh, Cigarette Smoke Exposure, and Atopy," Journal of the American Medical Association 249(8): 1022-1025, 1983.

21. Murray, A. and Morrison, B., "Passive Smoking and the Seasonal Difference of Severity of Asthma in Children," Chest 94(4): 701-708, 1988.

22. Neuspiel, D., Rush, D., Butler, N.R., Golding, J., Bijur, P.E. and Kurzon, M., "Parental Smoking and Post-Infancy Wheezing in Children: A Prospective Cohort Study," American Journal of Public Health 79(2): 168-171, 1989.

23. Ogston, S., Florey, C.D.V. and Walker, C.H.M., "Association of Infant Alimentary and Respiratory Illness with Parental Smoking and Other Environmental Factors," Journal of Epidemiology and Community Health 41(1): 21-25, 1987.

24. Schenker, M., Samet, J.M. and Speizer, F.E., "Risk Factors for Childhood Respiratory Disease: The Effect of Host Factors and Home Environmental Exposures," American Review of Respiratory Disease 128: 1038-1043, 1983.

25. Somerville, S., Rona, R.J. and Chinn, S., "Passive Smoking and Respiratory Conditions in Primary School Children," Journal of Epidemiology and Community Health 42(2): 105-110, 1988.

26. Strachan, D., Jarvis, M.J. and Feyerabend, C., "Passive Smoking, Salivary Cotinine Concentrations, and Middle Ear Effusion in 7 Year Old Children," British Medical Journal 298: 1549-1552, 1989.

27. Tsimoyianis, G., Jacobson, M.S., Feldman, J.G., Antonio-Santiago, M.T., Clutario, B.C., Nussbaum, M. and Schenker, I.R., "Reduction in Pulmonary Function and Increased Frequency of Cough Associated With Passive Smoking in Teenage Athletes," Pediatrics 80(1): 32-36, 1987.

28. Ware, J., Dockery, D.W., Spiro, A., Speizer, F.E. and Ferris, B.G., "Passive Smoking, Gas Cooking, and Respiratory Health of Children Living in Six Cities," American Review of Respiratory Disease 129: 366-374, 1984.

29. Ugnat, A.M., Mao, Y., Miller, A.B. and Wigle, D.T., "Effects of Residential Exposure to Environmental Tobacco Smoke on Canadian Children," Canadian Journal of Public Health 81(5): 345-349, 1990.

30. Greenberg, R.A., Bauman, K.E., Glover, L.H., Strecher, V.J., Kleinbaum, D.G., Haley, N.J., Stedman, H.C., Fowler, M.G. and Loda, F.A., "Ecology of Passive Smoking by Young Infants," Journal of Pediatrics 114: 774-780, 1989.

31. Berg, A.T., Shapiro, E.D. and Capobianco, L.A., "Group Day Care and the Risk of Serious Infectious Illnesses," American Journal of Epidemiology 133(2): 154-163, 1991.

32. Dijkstra, L., Houthuijs, D., Brunekreef, B., Akkerman, I. and Boleij, J.S.M., "Respiratory Health Effects of the Indoor Environment in a Population of Dutch Children," American Review of Respiratory Disease 142: 1172-1178, 1990.

33. McConnochie, K.M. and Roghmann, K.J., "Wheezing at 8 and 13 Years: Changing Importance of Bronchiolitis and Passive Smoking," Pediatric Pulmonology 6(3): 138-146, 1989.

34. Woodward, A., Douglas, R.M., Graham, N.M. and Miles, H., "Acute Respiratory Illness in Adelaide Children: Breast Feeding Modifies the Effect of Passive Smoking," Journal of Epidemiology and Community Health 44: 224-230, 1990.

35. Stern, B., Raizenne, M. and Burnett, R., "Respiratory Effects of Early Childhood Exposure To Passive Smoke," Environment International 15: 29-34, 1989.

36. Schilling, R., Letai, A.D., Hui, S.L., Beck, G.J., Schoenberg, J.B. and Bouhuys, A., "Lung Function, Respiratory Disease, and Smoking in Families," American Journal of Epidemiology 106(4): 274-283, 1977.

37. Binder, R., Mitchell, C.A., Hosein, H.R. and Bouhuys, A., "Importance of the Indoor Environment in Air Pollution Exposure," Archives of Environmental Health 31(6): 277-279, 1976.

38. Bouhuys, A., Lung Diseases in Rural and Urban Communities, Report Number HR-42912-1F, Sponsored by the Division of Lung Diseases, National Heart, Lung and Blood Institute, U.S. National Institutes of Health, Bethesda, Maryland, 77-89, June 10, 1977.

39. Gardner, G., Frank, A.L. and Taber, L.H., "Effects of Social and Family Factors on Viral Respiratory Infection and Illness in the First Year of Life," Journal of Epidemiology and Community Health 38: 42-48, 1984.

40. Harrington, W. and Krupnick, A., "Short-Term Nitrogen Dioxide Exposure and Acute Respiratory Disease in Children," Journal of the Air Pollution Control Association 35: 1061-1067, 1985.

41. Holma, B. and Winding, O., "Housing, Hygiene, and Health: A Study in Old Residential Areas in Copenhagen," Archives of Environmental Health 32(2): 86-93, 1977.

42. Kallail, K., Rainbolt, H.R. and Bruntzel, M.D., "Passive Smoking and Middle Ear Problems in Kansas Public School

Children," Journal of Communicable Disorders 20: 187-196, 1987.

43. Kerrebijn, J., Hoogeveen-Schroot, H.C.A. and van der Wal, M.C., "Chronic Nonspecific Respiratory Disease in Children, A Five Year Follow-Up Study," Acta Paediatr Scand Suppl. 261: 11-72, 1977.

44. Lebowitz, M. and Burrows, B., "Respiratory Symptoms Related to Smoking Habits of Family Adults," Chest 69(1): 48-50, 1976.

45. Love, G., Lan, S.P., Shy, C.M. and Struba, R.J., "The Incidence and Severity of Acute Respiratory Illness in Families Exposed to Different Levels of Air Pollution, New York Metropolitan Area, 1971-72," Archives of Environmental Health 36(2): 66-73, 1981.

46. Martin, C., Platt, S.D. and Hunt, S.M., "Housing Conditions and Ill Health," British Medical Journal 294: 1125-1127, 1987.

47. Salzman, M., Biller, H.F. and Schechter, C.B., "Passive Smoking and Croup," Archives of Otolaryngology and Head Neck Surgery 113: 866-868, 1987.

48. Shy, C., Creason, J.P., Pearlman, M.E., McClain, K.E., Benson, F.B. and Young, M.M., "The Chattanooga School Children Study: Effects of Community Exposure to Nitrogen Dioxide. II. Incidence of Acute Respiratory Illness," Journal of the Air Pollution Control Association 20(9): 582-588, 1970.

49. Harsten, G., Prellner, K., Heldrup, J., Kalm, O. and Kornfalt, R., "Acute Respiratory Tract Infections in Children: A Three-Year Follow-up from Birth," Acta Paediatr Scand 79(4): 402-409, 1990.

50. Thorton, A.J., Morley, C.J., Hewson, P.H., Cole, T.J., Fowler, M.A. and Tunnacliffe, J.M., "Symptoms in 298 Infants Under 6 Months Old, Seen at Home," Archives of Disease in Childhood 65(3): 280-285, 1990.

51. Victora, C.G., Smith, P.G., Barros, F.C., Vaughan, J.P. and Fuchs, S.C., "Risk Factors for Deaths Due to Respiratory Infections Among Brazilian Infants," International Journal of Epidemiology 18(4): 918-925, 1989.

52. Berkey, C., Ware, J.H., Dockery, D.W., Ferris, B.G. and Speizer, F.E., "Indoor Air Pollution and Pulmonary Function Growth in Preadolescent Children," American Journal of Epidemiology 123(2): 250-260, 1986.

53. Chen, Y. and Li, W., "The Effect of Passive Smoking on Children's Pulmonary Function in Shanghai," American Journal of Public Health 76(5): 515-518, 1986.

54. Corbo, G., Foresi, A., Valente, S. and Bustacchini, S., "Maternal Smoking and Bronchial Responsiveness in Children," American Review of Respiratory Disease 137(1): 245, 1987.

55. Hasselblad, V., Humble, C.G., Graham, M.G. and Anderson, H.S., "Indoor Environmental Determinants of Lung Function in Children," American Review of Respiratory Disease 123(5): 479-485, 1981.

56. Kauffmann, F., Tager, I.B., Munoz, A. and Speizer, F.E., "Familial Factors Related to Lung Function in Children Aged 6-10 Years," American Journal of Epidemiology 129(6): 1289-1299, 1989.

57. Masi, M., Hanley, J.A., Ernst, P. and Becklake, M.R., "Environmental Exposure to Tobacco Smoke and Lung Function in Young Adults," American Review of Respiratory Disease 138: 296-299, 1988.

58. Tager, I., Munoz, A., Rosner, B., Weiss, S.T., Carey, V. and Speizer, F.E., "Effect of Cigarette Smoking on the Pulmonary Function of Children and Adolescents," American Review of Respiratory Disease 131(5): 752-759, 1985.

59. Tager, I., Weiss, S.T., Munoz, A., Rosner, B. and Speizer, F.E., "Longitudinal Study of the Effects of Maternal Smoking on Pulmonary Function in Children," New England Journal of Medicine 309(120): 699-703, 1983.

60. Tashkin, D., Clark, V.A., Simmons, M., Reems, C., Coulson, A.H., Bourque, L.B., Sayre, J.W., Detels, R. and Rokaw, S., "The UCLA Population Studies of Chronic Obstructive Respiratory Disease. VII. Relationship Between Parental Smoking and Children's Lung Function," American Review of Respiratory Disease 129: 891-897, 1984.

61. Dodge, R., "The Effects of Indoor Pollution on Arizona Children," Archives of Environmental Health 37(3): 151-155, 1982.

62. Hosein, R. and Corey, P., "Multivariate Analysis of Nine Indoor Factors on FEV_1 of Caucasian Children," American Thoracic Society Meeting, Miami Beach, Florida, May 20-23, 1984. Poster Session #32.

63. Mandi, A., Galambos, E., Galgoczy, G., Szabo, M. and Kollar, K., "Relationship Between Lung Function Values and Air Pollution Data in Budapest School Children," Pneumonologie 150(2/4): 217-225, 1974.

64. Witorsch, R. and Witorsch, P., "Maternal Smoking and Pulmonary Performance in Children: A Critical Analysis," Indoor Air '87, Proceedings of the 4th International Conference on Indoor Air Quality and Climate, Berlin, 175-179, August 17-21, 1987.

65. Lebowitz, M., "The Effects of Environmental Tobacco Smoke Exposure and Gas Stoves on Daily Peak Flow Rates in Asthmatic and Non-Asthmatic Families." In: ETS-Environmental Tobacco Smoke: Report from a Workshop on Effects and Exposure Levels. R. Rylander, Y. Peterson and M.-C. Snella (eds.). Published simultaneously in European Journal of Respiratory Diseases, Supplement No. 133, Vol. 65, 90-97, 1984.

66. Lebowitz, M., Knudson, R.J. and Burrows, B., "Family Aggregation of Pulmonary Function Measurements," American Review of Respiratory Disease 129: 8-11, 1984.

67. Lebowitz, M., Holberg, C.J., Boyer, B. and Hayes, C., "Respiratory Symptoms and Peak Flow Associated with Indoor and Outdoor Air Pollutants in the Southwest," Journal of the Air Pollution Control Association 35: 1154-1158, 1985.

68. Melia, R., Florey, C.D.V., Altman, D.G. and Swan, A.V., "Association Between Gas Cooking and Respiratory Disease in Children," British Medical Journal II: 149-152, 1977.

69. Melia, R., Florey, C.D.V., Morris, R.W., Goldstein, B.D., John, H.H., Clark, D., Craighead, I.B. and Mackinlay, J.C., "Childhood Respiratory Illness and the Home Environment II. Association Between Respiratory Illness and Nitrogen Dioxide, Temperature and Relative Humidity," International Journal of Epidemiology 11(2): 164-169, 1982.

70. Melia, R., Chinn, S. and Rona, R.J., "Respiratory Illness and Home Environment of Ethnic Groups," British Medical Journal 296: 1438-1441, 1988.

71. Melia, R., Florey, C.D.V. and Chinn, S., "The Relation Between Respiratory Illness in Primary School Children and the Use of Gas for Cooking. I -- Results from a National Survey," International Journal of Epidemiology 8(4): 333-338, 1979.

72. Speizer, F., Ferris, B., Bishop, Y.M.M. and Spengler, J., "Respiratory Disease Rates and Pulmonary Function in Children

Associated with NO_2 Exposure," American Review of Respiratory Disease 121: 3-10, 1980.

73. Jedrychowski, W., Khogali, M. and Karim, M.A., "Height and Lung Function in Preadolescent Children of Kuwaitis and European Origin. The Pilot Survey on Health Effect of Gas Cooking in the Middle-East." In: Indoor Air Quality and Ventilation. London, Selper Ltd., Volume 2, 175-181, 1990.

74. Brunekreef, B., Houthuijs, D., Dijkstra, L. and Soleij, J.S.M., "Indoor Nitrogen Dioxide Exposure and Children's Pulmonary Function," Journal of the Air and Waste Management Association 40(9): 1252-1256, 1990.

75. Koo, L.C., Ho, J.H.C., Matsuki, H., Shimizu, H., Mori, T. and Tominaga, S., "A Comparison of the Prevalence of Respiratory Illnesses Among Nonsmoking Mothers and Their Children in Japan and Hong Kong," American Review of Respiratory Disease 138: 290-295, 1988.

76. Strachan, D. and Elton, R., "Relationship Between Respiratory Morbidity in Children and the Home Environment," Family Practice 3(3): 137-142, 1986.

77. Strachan, D., "Damp Housing and Childhood Asthma: Validation of Reporting of Symptoms," British Medical Journal 297: 1223-1226, 1988.

78. Platt, S.D., Martin, C.J., Hunt, S.M. and Lewis, C.W., "Damp Housing, Mould Growth, and Symptomatic Health State," British Medical Journal 298: 1673-1678, 1989.

79. Brunekreef, B., Dockery, D., Speizer, F.E., Ware, J.H., Spengler, J.D. and Ferris, B.G., "Home Dampness and Respiratory Morbidity in Children," American Review of Respiratory Disease 140: 1363-1367, 1989.

80. Watkins, C., Sittampalam, Y., Morrell, D.C., Leeder, S.R. and Tritton, E., "Patterns of Respiratory Illness in the First Year of Life," British Medical Journal 293: 794-796, 1986.

81. Kerigan, A., Goldsmith, C.H. and Pengelly, L.D., "A Three-Year Cohort Study of the Role of Environmental Factors in the Respiratory Health of Children in Hamilton, Ontario: Epidemiologic Survey, Design, Methods, and Description of Cohort," American Review of Respiratory Disease 133: 987-993, 1986.

82. Goren, A. and Hellmann, S., "Prevalence of Respiratory Symptoms and Diseases in Schoolchildren Living in a Polluted and in a Low Polluted Area in Israel," Environmental Research 45(1): 28-37, 1988.

83. Pope, C., "Respiratory Disease Associated with Community Air Pollution and a Steel Mill, Utah Valley," American Journal of Public Health 79(5): 623-628, 1989.

84. Frischer, T., Studnicka, M., Hesse, P. and Neumann, M., "Effects of Traffic Emissions on the Airways in Children," Pneumologie 44: 890-891, 1990.

85. Rutishauser, M., Ackermann, U., Braun, C., Gnehm, H.P. and Wanner, H.U., "Significant Association between Outdoor NO_2 and Respiratory Symptoms in Preschool Children," Lung, Supplement 168: 347-352, 1990.

86. Fleming, D., Cochi, S.L., Hightower, A.W. and Broome, C.V., "Childhood Upper Respiratory Tract Infections: To What Degree is Incidence Affected by Day Care Attendance?," Pediatrics 79(1): 55-60, 1987.

87. Hurwitz, E.S., Gunn, W.J., Pinsky, P.F. and Schonberger, L.B., "Risk of Respiratory Illness Associated with Day-Care Attendance: A Nationwide Study," Pediatrics 87(1): 62-69, 1991.

88. Wright, A.L., Holberg, C.J., Martinez, F.D., Morgan, W.J. and Taussig, L.M., "Breast Feeding and Lower Respiratory Tract Illness in the First Year of Life," British Medical Journal 299: 946-949, 1989.

89. Berwick, M., Zagraniski, R.T., Leaderer, B.P. and Stolwijk, J.A., "Respiratory Illness in Children Exposed to Unvented Combustion Sources." In: Indoor Air. Volume 2: Radon, Passive Smoking, Particulates and Housing Epidemiology. B. Berglund, T. Lindvall and J. Sundell (eds.). Stockholm, Swedish Council for Building Research, 255-260, 1984.

90. Osborne, J. and Honicky, R., "Health Effects of Heating With Wood: Chest Illness in Young Children and Indoor Heating with Woodburning Stoves." In: IAQ '89. The Human Equation: Health and Comfort. American Society of Heating, Refrigeration and Air-Conditioning Engineers, and the Society for Occcupational and Environmental Health, San Diego, California, April 17-20, 1989. Abstract.

91. Morris, K., Morganlander, M., Coulehan, J.L., Gahagen, S. and Arena, V.C., "Wood-Burning Stoves and Lower Respiratory Tract Infection in American Indian Children," American Journal of Diseases in Childhood 144: 105-108, 1990.

92. Nordvall, S., Eriksson, M., Rylander, E. and Schwartz, B., "Sensitization of Children in the Stockholm Area to House Dust Mites," Acta Paediatr Scand 77: 716-720, 1988.

93. Rylander,R., Sorensen, S., Goto,- H., Yuasa, K. and Tanaka, S., "The Importance of Endotoxin and Glucan for Symptoms in Sick Buildings." In: Indoor Air Quality: Symposium. Buenos Aires, National Academy of Sciences of Buenos Aires, 157-164, 1989.

94. Kort, H., van Bronswijk, J. and Schober, G., "Moulds, Mites and Moisture: A Preliminary Report on Six Cases of Fungal Damage in Dwellings." In: Present and Future of Indoor Air Quality. C. Bieva, Y. Courtois, M. Govaerts (eds.). Amsterdam, Excerpta Medica, 389-394, 1989.

95. Ownby, D. and McCullough, J., "Passive Exposure to Cigarette Smoke Does Not Increase Allergic Sensitization in Children," Journal of Allergy and Clinical Immunology 82(4): 634-638, 1988.

96. Johnson, C., Ownby, D., Stauchman, C., Blocki, S., Birg, G., Jacobsen, G. and McCullough, J., "Determinants of Immunoglobulin E (IgE) at Birth," American Journal of Epidemiology 132(4): 773-774, 1990.

97. Tager, I.B., "Health Effects of 'Passive Smoking' in Children," Chest 96(5): 1161-1164, 1989.

98. Sneddon, J.M. and Bearpark, T.M., "Indoor Air, Respiratory Health and Pulmonary Function in Children: A Critical Review." In: Indoor Air Quality and Ventilation. London, Selper Ltd., Volume 2, 143-154, 1990.

99. Witorsch, R.J. and Witorsch, P., "A Critical Analysis of the Relationship Between Parental Smoking and Pulmonary Performance in Children," Off Gesundh-Wes 51: 78-83, 1989.

100. Rubin, D. and Damus, K., "The Relationship Between Passive Smoking and Child Health: Methodologic Criteria Applied to Prior Studies," Yale Journal of Biological Medicine 61(5): 401-411, 1988.

Response to Question 2a(iii) (continued):

Asthma

Asthmatics are sometimes believed to be particularly vulnerable to various environmental influences, including PTS, but the scientific data in this area are contradictory and inconclusive. Most of these studies have been conducted neither in the home nor in the workplace but are clinical studies that have potential applicability to either setting. There are ten major studies on the possible association of exposure to PTS and acute respiratory symptoms in adult nonsmokers.[1-10] The studies are inconsistent in their reported results, and the authors often fail to adequately control for confounding factors. Furthermore, the authors often cannot rule out the possibility of psychological factors playing a role in the reported reactions of asthmatics to PTS.

A Discussion of the Published Studies

A 1981 study by Dahms, et al., reported decreases in the pulmonary function of several asthmatics exposed to environmental tobacco smoke.[1] However, the study suffers from several obvious limitations, including the unrealistic conditions under which the subjects were exposed to PTS; they were challenged with high levels of smoke in an enclosed smog chamber. In addition, as the authors themselves noted, their experiment lacked proper controls, and the

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effects observed may have been due to psychological, not physical, factors.

Contrary to Dahms, et al., a Canadian group, Pimm, et al., observed no systemic lung changes among asthmatics exposed to levels of tobacco smoke typically found in public places.[2] Later research by the co-authors of this study supports this conclusion.[3] They reported that respiratory data collected from a group of asthmatic volunteers exposed to tobacco smoke "do not suggest that asthmatic subjects have an unusual sensitivity" to such exposure. Although several volunteers claimed to have experienced wheezing and tightness in the chest due to the exposure, the researchers determined that the "physiological data give little support to the concept of a subgroup with particular sensitivity." They noted that these reactions probably were due to the "suggestibility" of the subjects.

The difficulty in determining the impact of psychological responses in such studies is clearly demonstrated by the results of two reports from Australia. Although a 1985 report by Knight and Breslin suggests that short-term PTS exposures are capable of inducing reactions in asthmatics,[4] an earlier study co-authored by Breslin tends to support the theory that psychological reactions may partially explain symptoms observed during such exposures.[5] In that study, Breslin and Ing reported that although asthmatics

exposed to tobacco smoke complained of subjective symptoms, no significant objective evidence of airways obstruction was observed.

Other studies also fail to provide support for claims regarding a relationship between PTS exposure and reactions in asthmatics. For example, Wiedemann, et al., at Yale University, examined the possible short-term effects of PTS exposure on a group of young asthmatic patients and observed no changes in lung flow rates.[6] They concluded that such exposures present "no acute respiratory risk" to asthmatics.

Tulane University scientists, Stankus, et al., recently assessed the effects of heavy exposure to PTS in a group of self-reported "tobacco smoke-sensitive" asthmatics.[7] They reported that two-thirds of the subjects did not experience significant changes in pulmonary function even after heavy, prolonged exposure to PTS, and that there was no association between reactions to PTS and hypersensitivity to tobacco leaf extract, which is commonly used in allergy testing.

A report by Lebowitz on data from a large-scale epidemiological study in the U.S. suggests that PTS in the home does not affect symptoms or pulmonary function in either children or adult asthmatics, but that dust and pollen in the home apparently can provoke such effects.[8] Another group of U.S. researchers,

Bailey, et al., also recently reported that exposure to PTS did not impair lung function in 263 asthmatic adult subjects.[9] A German research group, led by Magnussen, reported that one hour of exposure to PTS with 20 ppm carbon monoxide did not provoke acute respiratory symptoms or changes in lung function in 18 asthmatic adult subjects.[10]

Conclusion

The studies available on the reported association between adult asthma and exposure to PTS are inconclusive. The studies reporting an association suffer from several methodological problems. First, confounding factors are not adequately controlled for in many of the studies. Second, some of the studies report on inadequate sample sizes. Third, psychological factors have not been ruled out. Finally, a few of the clinical studies rely on unrealistic exposure conditions in enclosed smog chambers.

REFERENCES

1. Dahms, T.E., Bolin, J.F. and Slavin, R.G., "Passive Smoking Effects on Bronchial Asthma," Chest 80(5): 530-534, 1981.

2. Pimm, P.E., Shephard, R.J. and Silverman, F., "Physiological Effects of Acute Passive Exposure to Cigarette Smoke in Asthmatics," Federal Proceedings 36(3): 606, 1977. Abstract.

3. Shephard, R.J., Collins, R. and Silverman, F., "'Passive Exposure of Asthmatic Subjects to Cigarette Smoke," Environmental Research 20: 392-402, 1979.

4. Knight, A. and Breslin, A.B.X., "Passive Cigarette Smoking and Patients with Asthma," The Medical Journal of Australia 142: 194-195, 1985.

5. Ing, A. and Breslin, A.B.X., "The Effect of Passive Cigarette Smoking on Asthmatic Patients," Proceedings, The Thoracic Society of Australia, 543, October, 1983. Abstract.

6. Wiedemann, H.P., Mahler, D.A., Loke, J., Virgulto, J.A., Snyder, P. and Matthay, R.A., "Acute Effects of Passive Smoking on Lung Function and Airway Reactivity in Asthmatic Subjects," Chest 89(2): 180-184, 1986.

7. Stankus, R.P., Menon, P.K., Rando, R.J., Glindmeyer, H., Salvaggio, J.E. and Lehrer, S.B., "Cigarette Smoke-Sensitive Asthma: Challenge Studies," Journal of Allergy and Clinical Immunology 82: 331-338, 1988.

8. Lebowitz, M., "The Effects of Environmental Tobacco Smoke Exposure and Gas Stoves on Daily Peak Flow Rates in Asthmatic and Non-Asthmatic Families." In: ETS-Environmental Tobacco Smoke: Report from a Workshop on Effects and Exposure Levels. R. Rylander, Y. Peterson and M.-C. Snella (eds.). Published simultaneously in European Journal of Respiratory Diseases, Supplement No. 133, Vol. 65, 90-97, 1984.

9. Bailey, W.C., Richards, J.M., Manzella, B.A., Brooks, C.M., Windsor, R.A. and Soong, S.J., "Characteristics and Correlates of Asthma in a University Clinic Population," Chest 98(4): 821-828, 1990.

10. Magnussen, H., "The Effect of Passive Smoking on Airway Tone and Responsiveness in Children and Adults with Bronchial Asthma," The Toxicology Forum: 1990 European Meeting, Budapest, Hungary. June 18-22, 1990: 202-205.

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Response to Question 2a(iii) (continued):

Allergy

Although some individuals are annoyed by the sight and smell of tobacco smoke and a few even report experiencing irritation, the existence of human allergens in tobacco smoke has not been established scientifically.

A number of research groups have been unable to conclude that humans actually experience a true tobacco smoke allergy.[1-6] In 1980, for example, a group of researchers noted that "direct evidence that tobacco smoke is immunogenic [capable of evoking a specific response] in man is yet to be documented."[5] A more recent report by this same group affirms this conclusion.[6]

Claims about a human tobacco allergy stem primarily from studies in which tobacco leaf extract has been reported to cause allergic skin responses in some people, usually in those who already experience allergic reactions to other substances such as weeds.[7-9] However, as an English immunologist pointed out, there are "great difficulties" in determining whether positive reactions to tobacco leaf extracts are relevant to clinical responses to tobacco smoke.[4] Although he noted that there may be substances in tobacco smoke which could "theoretically" act as such agents, he concluded that

"there is no proof that the specific sensitization to tobacco smoke exists."

It has also been hypothesized that tobacco smoke is capable of provoking an asthma attack as an allergic reaction.[10-11] However, a Swedish specialist concluded that such results are not proof of a tobacco allergy because the studies, which used tobacco extracts, did not differentiate between non-specific and true allergic reactions in evaluating the results of skin tests and bronchial provocation.[12] Consequently, he stated, "for the present, the question as to whether allergy to cigarette smoke exists or not should be kept open." Other research affirms this point. Scientists reported in 1988 that tobacco leaf sensitivity was not associated with decreased pulmonary function in allergic asthmatics.[13]

Certainly, there appear to be people who may be sensitive to tobacco smoke, but personal annoyance and emotional reactions should not be confused with genuine allergic reactions. In many cases, the individual may be responding to high room temperatures, lack of ventilation, or even to the mere sight of tobacco smoke. Indeed, Dr. John Salvaggio, the director of an allergic disease center in the U.S., has suggested that reported reactions to tobacco smoke may be irritative rather than allergic. After reviewing the studies on the allergy question, he concluded that "there is no

proof that tobacco smoke is allergenic in man."[14]

Accordingly, it is not surprising that researchers at the Mayo Clinic failed to find any evidence of tobacco smoke allergies in their tests of subjects who considered themselves allergic to tobacco and tobacco smoke.[15] It has been reported in other studies that people who claim to be "smoke sensitive" did not react more frequently to tobacco leaf or smoke extract than those who are "smoke resistant."[13,16]

On the basis of these data, it must be questioned what people really mean when they say they are "allergic" to tobacco smoke. It may be that they simply do not like the sight and smell of tobacco smoke and are interpreting their reaction to mean that they are "allergic" to environmental tobacco smoke. But, as has been pointed out, such personal reactions should <u>not</u> be regarded as true tobacco smoke allergies.

REFERENCES

1. Justus, D. and Adams, D., "Evaluation of Tobacco Hypersensitivity Responses in the Mouse: A Potential Animal Model for Critical Study of Tobacco Allergy," Int Arch Allergy Appl Immunol 51(6): 687-695, 1976.

2. Lehrer, S., et al., "Immunochemical Properties of Tobacco Smoke and Leaf Components," Fed Proc 37(6): 1456, 1978.

3. Lehrer, S., et al., "Immunogenic Properties of Tobacco Smoke," J Allerg Clin Immunol 62(6): 368-370, 1978.

4. Taylor, G., "Tobacco Smoke Allergy -- Does It Exist?" In: Environmental Tobacco Smoke Effects on the Nonsmoker: Report from a Workshop. R. Rylander (ed.). Published in Scand J Respir Dis Supplement 91: 50-55, 1974.

5. Lehrer, S., et al., "Immunogenicity of Tobacco Smoke Components in Rabbits and Mice," Int Arch Allergy Appl Immunol 62: 16-22, 1980.

6. Lehrer, S., et al., "Tobacco Smoke Sensitivity: A Result of Allergy?," Ann Allergy 56: 1-10, 1986.

7. Gleich, G., et al., "Allergy to Tobacco Antigens," J Allerg Clin Immunol 63(3): 211-212, 1979.

8. Harkavy, J., "Tobacco Allergy in Cardiovascular Disease: A Review," Ann Allerg 26: 447-459, 1968.

9. Speer, F., "Tobacco and the Nonsmoker: A Study of Subjective Symptoms," Arch Environ Health 16(3): 443-446, 1968.

10. Stahle, I. and Tibbling, L., "Tobaksallergi hos Patienter Med Asthma Bronchiale (Tobacco Allergy Among Patients with Bronchial Asthma)," Lakartidningen 75(17): 1711-1713, 1978. Translation.

11. Tibbling, L., "Elicitation of Bronchial Asthma with Tobacco Extract." In: Abstracts of the 4th World Conference on Smoking and Health. Stockholm, Sweden, June 18-21, 1979.

12. Bylin, G., "Tobaksallergi -- finns den? (Tobacco Allergy -- Does It Exist?)," Lakartidningen 77(16): 1530-1532, 1980. Translation.

13. Stankus, R. and Lehrer, S., "Inhalation Challenge Studies on Tobacco Smoke Sensitive Asthmatics." In: Indoor and Ambient

Air Quality. R. Perry and P. Kirk (eds.). London, Selper Ltd., 303-304, 1988.

14. Salvaggio, J., Statement, U.S. Congress Committee on Agriculture, Subcommittee on Tobacco, Effect of Smoking on Non-smokers, Hearing, 95th Congress, 2nd Session, Washington, D.C., U.S. Government Printing Office, 46-49, September 7, 1978.

15. McDougall, J. and Gleich, G., "Tobacco Allergy -- Fact or Fancy?," J Allerg Clin Immunol 57(3): 237, 1976. Abstract.

16. Lehrer, S., et al., "Immunological Evaluation of Tobacco Smoke 'Sensitive' Individuals by Skin, Radioallergosorbent and Precipitin Tests," J Allerg Clin Immunol 67: 49, 1981. Abstract.

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PHILIP MORRIS RESPONSE TO OSHA RFI

Question 2a(iv): "What data are available that associate adverse health effects with exposure to radon?"

Summary:

Radon, a naturally occurring radioactive gas, is ubiquitous in the indoor environment. Epidemiologic studies report an increased risk of lung cancer associated with the inhalation of radon and its radioactive "daughters" in underground miners. Although questions remain about radon dosimetry, it has been suggested that radon may be associated with an increased risk of lung cancer in the general population. Radon levels in buildings may be reduced with adequate ventilation.

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PHILIP MORRIS RESPONSE TO OSHA RFI

Question 2a(iv): **"What data are available that associate adverse health effects with exposure to radon?"**

Response:

Epidemiologic studies report that the inhalation of radon progeny is associated with an increased risk of lung cancer among underground miners.[1,2] Because elevated radon levels have been measured in some residences, it has also been suggested that radon exposure may be responsible for between ten and 20 percent of the attributable lifetime lung cancer risk in the general population.[3,4]

Radon-222 is a naturally occurring radioactive gas, ubiquitous in both outdoor and indoor environments, produced by the radioactive decay (breakdown) of uranium; radon itself decays, producing "progeny" or "daughters," which, along with radon itself, may be inhaled.[2] Some of the progeny, namely the isotopes polonium-214 and -218, have short half-lives, meaning that they may decay while in the lungs, emitting alpha particles that then impact on lung tissue.[5] Radon progeny have been described as the most important source of natural irradiation in the general population.[3,6]

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Questions remain about radon dosimetry and lung cancer risk.[2] For instance, some have claimed that radon exposure and cigarette smoking interact to increase the risk of lung cancer.[7] Models of the purported interaction include multiplicative (synergistic), additive and submultiplicative models. However, Jacobi and Paretzke, using a modified proportional hazard model, adapted to account for relevant epidemiological data, concluded that the relative fractions of radon-related lung cancer rates could be nearly the same for smokers and nonsmokers.[8]

With regard to epidemiologic studies, Harley and Pasternack wrote that[9]

> [t]he problem of attributable risk where smoking and radon daughter exposures are combined cannot be resolved at present. . . One recent study of Swedish iron miners which has nearly complete follow-up to death shows evidence that the attributable risk in smoking miners is about the same as in nonsmoking miners. That is, the 'extra' risk from radon daughters is the same whether the individual smokes or not.

Elsewhere, Harley notes that in one miner study[10]

> Radford and Renard found that the excess lung-cancer rate in nonsmokers was as high as in smokers with equivalent radiation exposure. When the actual rate of lung cancer was compared with the expected rate, the ratio was higher in nonsmokers than in smokers, per unit of radiation exposure.

Furthermore, in a case-control study of 292 female lung cancer cases in the city of Stockholm, Sweden, Svensson and colleagues[11] reported a relative risk of 2.2 for lung cancer associated with "living in dwellings close to the ground in areas with an increased risk of radon emanation." Nevertheless, they also reported that smoking habits were <u>not</u> a major confounder of the reported association.

Moreover, at least one animal inhalation study reported that the incidence of lung tumors was less in beagle dogs exposed to cigarette smoke and radon progeny:[12]

> [C]igarette-smoke exposure, under the conditions of the experiment, had a mitigating effect on radon-daughter-induced respiratory cancer in dogs.

(Four groups of dogs were exposed as follows: to radon, radon daughters, and uranium ore dust; to radon, radon daughters, uranium ore dust and cigarette smoke; to cigarette smoke only; or to room air.) The authors proposed that increased mucus production in the smoking dogs was the best explanation for the small number of respiratory tumors in that group.[13]

With regard to "passive" tobacco smoke (PTS), it has been suggested that PTS can affect the dosimetry of indoor radon

by modifying the behavior of its decay products (progeny).[14] Because airborne particles (e.g., from smoke or dust) provide sites for attachment, their presence may reduce the "unattached" fraction of radon progeny, i.e., the free ions produced in the decay process. (The "attached" fraction consists of progeny present on larger (around 130 nm; 1 nm equals 1/1,000,000,000 of a meter, or approximately 4/100,000,000 of an inch) airborne particles.) Because the progeny in the "unattached" fraction are around 3 nm in size, they are thought to deposit more efficiently in the respiratory tract, particularly in the bronchial rather than the pulmonary regions. It has been estimated that the dose reduction when PTS is present in the air may be as much as 35%. Moreover,[15]

> [A] combination of many factors have to be considered when calculating the uptake and retention of various pollutants, and . . . airborne concentration information is simply insufficient to properly develop population risk estimates. Added to this, individual differences in physique, breathing pattern, deposition, clearance and metabolism may also occur.

In a recent study, Stebbings and Dignam measured total radon body burden (i.e., the level of external and internal radon contamination), and reported a "clear negative association between current smoking and the presence of high radon daughter contamination levels" in their study subjects [emphasis added].[16] The authors noted:

This does not directly support the relationship assumed in the current literature that passive smoking increases lung dose by increasing the equilibrium ratio. The latter may occur, but there may be stronger countervailing factors, such as reduction of superficial plateout.

Furthermore, the University of Pittsburgh Radon Project included over 73,000 radon measurements in homes and reported that radon levels tended to be lower in smokers' homes than in nonsmokers' homes.[17] The researchers suggested that smokers might tend to open windows more often (i.e., increasing ventilation), which could influence radon levels.

REFERENCES

1. Ellett, W.H. and Nelson, N.S., "Epidemiology and Risk Assessment: Testing Models for Radon-Induced Lung Cancer." In: Indoor Air and Human Health. R.B. Gammage and S.V. Kaye (eds.). Chelsea, Michigan, Lewis Publishers, Inc., 79-107, 1985.

2. Guilmette, R.A., Johnson, N.F., Newton, G.J., Thomassen, D.G. and Yeh, H.C., "Risks from Radon Progeny Exposure: What We Know, and What We Need to Know," Annual Review of Pharmacology and Toxicology 31: 569-601, 1991.

3. International Commission on Radiological Protection, "Lung Cancer Risk from Indoor Exposures to Radon Daughters," Acta Oncologica 26: 489-490, 1987.

4. Maher, E.F., Rudnick, S.N. and Moeller, D.W., "Effective Removal of Airborne 222Rn Decay Products Inside Buildings," Health Physics 53: 351-356, 1987.

5. Nero, A., "Earth, Air, Radon and Home," Physics Today 32-39, April 1989.

6. Hewitt, C.N. and Kelly, M., "Environmental Radon: A Brief Review," Environmental Technology 11: 387-392, 1990.

7. Samet, J.M. and Nero, A.V., "Indoor Radon and Lung Cancer," The New England Journal of Medicine 320: 591-594, 1989.

8. Jacobi, W. and Paretzke, H.G., "Risk Assessment for Indoor Exposure to Radon Daughters," The Science of the Total Environment 45: 551-562, 1985.

9. Harley, N.H. and Pasternack, B.S., "Response to 'Application of Epidemiologic Methods to the Study of Radon and Lung Cancer,'" Health Physics 42: 734-735, 1982.

10. Harley, N.H., "Radon and Lung Cancer in Mines and Homes," The New England Journal of Medicine 319: 1525-1527, 1984.

11. Svensson, C., Eklund, G. and Pershagen, G., "Indoor Exposure to Radon from the Ground and Bronchial Cancer in Women," International Archives of Occupational and Environmental Health 59: 123-131, 1987.

12. Cross, F.T., Palmer, R.F., Filipy, R.E., Dagle, G.E. and Stuart, B.O., "Carcinogenic Effects of Radon Daughters, Uranium

Ore Dust and Cigarette Smoke in Beagle Dogs," Health Physics 42: 33-52, 1982.

13. Gies, R.A., Cross, F.T. and Dagle, G.E., "A Histologic Study of the Influence of Cigarette Smoking in Suppressing Rn Daughter Carcinogenesis in Dogs," Health Physics 53: 327-329, 1987.

14. Pritchard, J.N. and Strong, J.C., "The Influence of Environmental Tobacco Smoke on Radon Dosimetry." In: Indoor Air '90: Proceedings of the Fifth International Conference on Indoor Air Quality and Climate. Toronto, Canada, Volume 3, 33-38, 1990.

15. McAughey, J.J., Pritchard, J.N. and Black, A., "Risk Assessment of Exposure to Indoor Air Pollutants," Environmental Technology 11: 295-302, 1990.

16. Stebbings, J.H. and Dignam, J.J., "Contamination of Individuals by Radon Daughters: A Preliminary Study," Archives of Environmental Health 43: 149-154, 1988.

17. Cohen, B.L. and Gromicko, N., "Variation of Radon Levels in U.S. Homes with Various Factors," JAPCA 38: 129-134, 1988.

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PHILIP MORRIS RESPONSE TO OSHA RFI

Question 2(d): "Based on observations in your workplace or your knowledge from other sources, how much lost work time and decreased productivity may be traceable to illnesses relating to poor indoor air quality? What is the basis for your estimate?"

Summary:

Several studies in the scientific literature on indoor air quality suggest that poor indoor air quality is related to lost work time and decreased productivity. Insofar as poor indoor air quality is associated with inadequate ventilation, the literature indicates that an increase in ventilation to rates specified in ASHRAE Standard 62-1989 may improve productivity and reduce absenteeism rates. The cost incurred in an increase in outdoor air ventilation is, according to these authors, offset by increased worker morale, productivity and reduced absenteeism.

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PHILIP MORRIS RESPONSE TO OSHA RFI

Question 2(d): "Based on observations in your workplace or your knowledge from other sources, how much lost work time and decreased productivity may be traceable to illnesses relating to poor indoor air quality? What is the basis for your estimate?"

<u>Response</u>:

A number of estimates and examples of lost work time and decreased productivity attributable to poor indoor air quality are found in the published literature. For example, Professor James Woods has estimated that a 25 percent energy savings in building operations (e.g., reduced ventilation) may be out-weighed by the loss of anywhere from 2 to 6 minutes per person per day of productive concentration.[1]

A 1990 report issued by the United States Air Force estimates the following:[2]

> The cost of personnel in the Air Force averages about $250.00 per square foot per year. The maintenance and energy costs for all operations in a building rarely exceed $7.00 per square foot per year. Of the $7.00, no more than $2.00 are energy costs. If the unlikely proved true and we saved half of all energy costs by running closed or minimum fresh air cycles, that would be $1.00/sq ft/yr. This is the equivalent of seven hours per square foot of personnel time. This is less than two minutes per day per year. If only two minutes per day

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productive time is lost, then all energy savings are wiped out. Respiratory disease is at least 1.5 to 3 times more likely in a tight environment. Common respiratory infection episodes last eight to ten days with one to two days of absence being usual. The average number of respiratory infections involving colds and flu is one per person per year based on National Center for Health Statistics data. Tight buildings are likely to raise that number to between 1.5 and 3.0 episodes per person per year. Using the lower figure of one day of loss per episode, we have raised the lost days from one to between 1.5 and 3 per person per year with an average of 2.25 days. This is an average increase of 1.25 days. By this measure alone we have lost 10 hours, three more than is required to put us in the deficit column. Three of the days of infection are likely to suffer from reduced productivity by at least 20% for a total of 60% of a day. Cost is now 1.85 excess days lost; 1.85/220 = 0.84% x $250/sq ft/yr = $2.10/sq ft/yr. This cost already exceeds the savings possible by running the air handler with inadequate fresh air by 2.1 times. This analysis does not begin to address the losses due to aggravation of pre-existing problems such as asthma and allergies, the cost of other diseases known to result from improper maintenance and operation of air handlers, the loss in productivity due to irritant effects of poorly controlled 'comfort' parameters or the losses due to the social atmosphere surrounding the problems generated. The actual savings from running closed cycle are even less than the generous $1.00 we allowed. It is usually necessary to increase total outside added air from roughly 10% of total flow to obtain adequate fresh air and control CO_2 to appropriate levels. If all of the $2.00 were going to climate control, this represents only 20 cents rather than a dollar in savings. Since this is so, the actual relative loss from increase in infections is $2.10/0.20 = 10.5 times the possible energy savings if all energy is used for heating. This is a poor bargain.

In testimony before the United States Congress in 1991, a representative from the Sheet Metal and Air Conditioning Contractors National Association, Inc., (SMACNA) reported on a study conducted in which building energy cost simulations at two levels of ventilation were evaluated. In the study, outside air intake was increased 50 percent and the estimated increase in energy cost was $1,800.00 on an annual basis. According to SMACNA, the same study "compared the cost of absent or sick workers affected by unhealthy ventilation and concluded that increased ventilation cost 1/135 of the cost of absent and sick employees."[3]

In another 1991 publication, Mr. Peter Binnie describes a hypothetical situation where energy costs for maintaining acceptable IAQ are considered in terms of employee absentee rates. He writes:[4]

> Consider the following situation. In a typical building of 100,000 square feet, the move from 20 cubic feet per minute (10 litres per second) of fresh air per person down to, say, 5 cubic feet per minute (2.5 litres per second) per person may on average result in cost savings of perhaps $20,000 per annum. This could represent a 40% savings in the energy costs associated with ventilation, or 10-15% of the building's overall energy budget. Everyone in upper management would be pleased, and one can be certain that future years' budgets would be reduced to reflect the actual costs of this first year. Thus, the building is now doomed to function with no more than 5 cubic feet per minute (2.5 litres per second) per person. As HBI, ASHRAE and many others have found, this

action results in unacceptable indoor pollution and increased absentee rates.

In the United States and Europe, offices are commonly staffed with a generous average of 150 square feet of space per employee. Thus, in the hypothetical building of 100,000 square feet, there would be 67 staff. If they are paid the minimum wage of $15,000 per annum, the payroll bill would reach $10 million per annum. This means that every 1% of absenteeism would cost $100,000 per annum. Surely a decision to save $20,000 per year on energy that results in reducing worker productivity and increasing absentee rates involving hundreds of thousands of dollars in costs is poor business judgment.

REFERENCES

1. Kreiss, K., "The Sick Building Syndrome: Where Is the Epidemiologic Basis?," AJPH 80(10): 1172-1173, 1990.

2. Carpenter, D. and Poitrast, B., "Recommended Carbon Dioxide and Relative Humidity Levels for Maintaining Acceptable Indoor Air Quality," USAF Occupational and Environmental Health Laboratory, Brooks AFB, Texas: October, 1990.

3. The Sheet Metal and Air Conditioning Contractors' National Association, Inc., Statement on HR 1066 to The House Science, Space and Technology Committee Environment Subcommittee, May 9, 1991.

4. Binnie, P., "The Role of Ventilation in Controlling the Quality of Indoor Air, Including ETS." In: Other People's Tobacco Smoke. A. Armitage (ed.). E. Yorks, U.K., Galen Press, 159-172, 1991.

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PHILIP MORRIS RESPONSE TO OSHA RFI

Question 3(a): "What correlation, if any, can be made between symptoms presented and IAQ complaints and type of causative agent? For example, are certain symptoms more indicative of exposure to chemical contaminants as opposed to biological contaminants? Please give examples.

(b) If such a correlation has been made, how effective is this information in identifying sources of contaminants?"

Summary:

Four large databases on sick-building syndrome investigations, including databases from NIOSH, Health and Welfare Canada, T.D. Sterling and Associates, Ltd., and Healthy Buildings International, do not reveal significant correlations between IAQ complaints and symptoms and specific types of causative agents. In over 50 percent of all sick-building cases, symptoms and complaints are abated by increasing ventilation to levels comparable to those specified in ASHRAE 62-1989. In addition, the four databases indicate that complaints and symptoms can be correlated to tobacco smoking in only two to four percent of all sick-building investigations.

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PHILIP MORRIS RESPONSE TO OSHA RFI

Question 3(a): "What correlation, if any, can be made between symptoms presented and IAQ complaints and type of causative agent? For example, are certain symptoms more indicative of exposure to chemical contaminants as opposed to biological contaminants? Please give examples.

(b) If such a correlation has been made, how effective is this information in identifying sources of contaminants?"

Response:

The scientific literature contains a number of studies on "sick building syndrome," a condition which gives rise to a number of complaints, including headaches, nausea, coughs, sore eyes and breathing difficulties. Research also indicates that this complex pattern of symptoms is commonly reported in modern office buildings whether or not smokers are present.[1,2,3,4,5,7,8]

The reported findings of several Canadian investigators demonstrate this point. In 1983, two of these researchers reported on their extensive review of over 150 indoor air quality evaluations of office buildings compiled by U.S. government agencies, universities and others.[5] After examining the data collected during these evaluations, they concluded that smoking did not significantly

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affect either indoor atmospheres or the frequency of worker complaints and symptoms:

> The review of available studies does not provide any objective evidence that either pollution levels or patterns of health related complaints differ in some remarkable way between locations with or without smoking restrictions.

They did observe that "inadequate" ventilation creates conditions "where discomfort and illness result irrespective of whether or not smoking is permitted." These observations were reaffirmed in reports published in 1987 and 1989.[4,6] In their 1989 report, the researchers noted that smoking was related to complaints in only 12 of 408 (less than three percent) of the building investigations included in the database.

Government investigators with Health and Welfare Canada (HWC), in their report on 94 building studies, also noted that only five percent of the complaints were attributable to indoor constituents such as photocopy machine emissions and PTS.[3] In a 1990 update, these investigators reported on data from a total of 1,362 building investigations. Inadequate ventilation was identified as a problem in 52 percent of the buildings.[7]

Similarly, researchers with the U.S. National Institute of Occupational Safety and Health (NIOSH), in a review of 203 air

quality investigations of government and business offices, schools and health care facilities, concluded that tobacco smoke played a contributing role in only four (two percent) of the building complaints investigated.[1] A large majority of the complaints were traced to general building contamination and inadequate ventilation. Recently, NIOSH officials reported on an additional 326 building investigations conducted by the agency through 1988.[8] Over one-half of the investigations revealed inadequate ventilation as the source of complaints.

In 1988, a representative of a U.S. firm specializing in the maintenance of office air conditioning and heating systems reported on 223 individual indoor air quality investigations of publicly and privately owned office buildings.[2] As in the NIOSH investigations, PTS was implicated in only four percent of the buildings investigated. He stated that the majority of indoor air quality problems in modern office buildings may be traced to inadequate fresh air circulation and to poorly maintained ventilation systems which act as breeding grounds for fungi, bacteria and other contaminants. He also suggested that visible tobacco smoke ought to be considered a symptom, rather than a cause, of general indoor air quality problems, in that PTS is often the only visible sign that a ventilation problem exists.

In 1990, investigators from the same firm reported on a survey of 26 commercial office buildings in 20 cities in Switzerland.[9] Unacceptably low ventilation rates were reported in more than half of the buildings investigated. PTS was associated with complaints in only six buildings, all of which also were affected by inadequate ventilation. Acceptable ventilation rates, according to the researchers, resulted in low levels of PTS constituents.

It is perhaps understandable, given the easy recognition of PTS, that persons experiencing sick building symptoms tend to blame PTS. Indeed, researchers have indicated that the mere visibility or presence of tobacco smoke may provoke claims that PTS is the cause of reported symptoms and complaints.[10,11] However, removal of PTS through smoking bans may serve only to divert attention from more basic, underlying indoor air quality problems. As one commentator pointed out: "Removing the smoker entirely, then, may not affect health and comfort problems in 95% to 98% of sick buildings."[12]

Other published studies in the literature tend to support conclusions from the four major databases on sick building syndrome. For example, in 1989, Hedge, et al., reported on an investigation of indoor air quality and health complaints in two office buildings with different ventilation systems.[13] While the researchers noted

that self-reports of exposure to PTS were associated with an increase in perceived symptom prevalence, they also observed that no significant correlations between PTS constituents, as measured in the office areas, and symptom prevalence could be found.

A 1991 report by Hawkins and Wang[14] ranked a number of variables related to self-reported symptoms of sick building syndrome. Those variables included (from "most frequently" to "least frequently" reported): "humidity," "satisfaction with work," "active smoking," "gender," "exposure to PTS," "office light," and "doing professional work." They concluded:

> Building Sickness Score was associated with many factors. Sick building syndrome symptoms are influenced by multiple variables of which the environmental factor of humidity and the psychological factors of work, sex, and occupation are important.

In another 1991 report, Hedge, et al., observed that "in an ongoing study of the effects of smoking policy on IAQ and SBS in air conditioned offices, no evidence of substantial IAQ differences have been found in nonsmoking open plan offices in 15 buildings with various kinds of restrictive smoking policies or 3 buildings that prohibit smoking. To date, 3,155 workers in these offices have been surveyed and results show that, contrary to expectations, workers in the smoking-prohibited buildings on average

report more symptoms than in those buildings with a restrictive smoking policy."[15]

The same investigators also noted that other research by Valbjorn, et al. in 1990 "found that samples from supply ducts in 13 ventilation systems contained from 70 to 6,200 viable fungi per gram of dust and from 50 to 5,000 viable bacteria per gram of dust. Samples from ventilation filters also contained 70 to 3,400 fungi per gram of dust and 100 to 6,700 viable bacterial per gram of dust. Dust in ventilation supply ducts was found to be similar to that sampled from the floor. Ghazi (1990) reports that removal of dust mite feces from carpets in an office resulted in a 25% reduction in SBS symptoms."[15]

In 1983, Sterling and co-workers reported on a number of sick building investigations in which symptoms were directly related to (1) detergent residues, (2) fibrous glass dust from duct work, (3) formaldehyde off-gassing from insulation, (4) photo-chemical smog formation and (5) viable organisms located in duct systems, cooling towers and humidification chambers.[16]

Indoor air quality in schools has recently become an issue of considerable concern. In 1989, Helsing and co-workers reported the results of an IAQ investigation in a school.[17] They reported that "there was an insufficient fresh air supply to some

classrooms, and a large percentage of students exhibited classic symptoms of Sick-Building Syndrome, i.e., headache, eye burning, fatigue. Correcting the ventilation problems resulted in reduction of symptoms to a level approximately equal to that of students in other schools in the county."

Similarly, investigations by Hanssen (1987)[18] and Beller (1989)[19] reported that low air exchange rates in combination with installation of new building materials was the main cause of complaints in the schools they investigated.

Norback and colleagues have published a number of investigations on sick building syndrome. In a 1987 report, the researchers compared groups of individuals reporting SBS symptoms and noted that while "SBS-symptoms were not uncommon among non-exposed reference groups, a remarkable over frequency of SBS-symptoms were demonstrated among the SBS-groups in this investigation."[20] Neither atopy (a predisposition toward the development of allergies) nor smoking habits could explain their observation. In a 1989 follow-up report, the researchers reported that "the sick building groups did not differ from the reference groups with regard to other factors such as mean age, smoking habits, atopy frequency, work stress, or work satisfaction."[21] In still another investigation, the research group reported on a sick building investigation in which the removal of wall-to-wall carpets

resulted in a decrease in reported systems to a level similar to those in individuals without previous or current exposure to carpeting.[22]

A 1989 report by Dr. James Woods assesses 30 cases of "problem buildings" investigated by the Honeywell Corporation since 1986.[23] Woods' research indicates that 75% of the buildings investigated had inadequate outdoor supply air intake. Similarly, 75% of the buildings had inadequate air distribution to occupied spaces, and 75% of the buildings suffered from inadequate maintenance protocols.

Kim (1990) summarized investigations of 105 problem buildings by Clayton Environmental Consultants. She wrote:[24]

> In a survey of 105 problem buildings, Clayton found that 53 percent had maintenance problems, 49 percent had operational problems (such as improper handling of control equipment), and 33 percent had design problems. Mechanical engineers evaluated the HVAC systems in 70 of the buildings, in which they found that 75 percent had maintenance problems, 70 percent had operational problems, and 47 percent had design problems. Of the 105 buildings, 95 were sampled for contaminants. Of these 28 were found to have problem levels of microbial contaminants, 26 had volatile organic compounds, and 13 had combustion products.

In 1987, Burge and co-workers reported on symptoms of building sickness among 4,373 office workers in 42 different office

buildings and under 47 different ventilation conditions in the United Kingdom.[25] They reported that the most common work-related symptoms were lethargy, blocked nose, dry throat and headache. These symptoms increased substantially once the supply air was either chilled or humidified. In a similar study in Denmark, Skov and colleagues examined sick building syndrome reports in 4,369 office workers.[26] Their research indicated that indoor climate perception was strongly related to the prevalence of SBS symptoms. Lifestyle factors were only weakly associated with the reporting of symptoms.

Canadian researchers recently reported results of a building investigation which included measurements of temperature, humidity, dust, nicotine, formaldehyde, volatile organic compounds and CO_2.[27] Occupants of the building complained of poor indoor air quality and questionnaire evaluation revealed a substantial number of complaints about unsatisfactory thermal comfort, dry air, drowsiness and eye irritation. However, all measured parameters, including temperature and humidity, were within accepted comfort and exposure guidelines. The authors were unable to correlate any single measured environmental parameter with complaints and symptoms. They concluded that their investigation "showed that complaints reported by the occupants were associated with perceived rather than measured levels of indoor environmental parameters."

In summary, reported exposures to PTS have been directly associated with sick building syndrome complaints in only 2 to 4 percent of the buildings investigated.[1-8] Because it is easily identifiable in the indoor environment, PTS is often blamed for IAQ problems. However, recent research by Winneke and colleagues suggests that an individual who is predisposed to annoyance from PTS will be more likely under experimental exposure conditions to respond with annoyance symptoms.[11] A NIOSH psychologist, Dr. Michael Colligan, offers an explanation for this kind of response. He writes:[28]

> It appears then, that the individual is sensitive to fluctuations in the functioning of the autonomic nervous system. When perceived changes in his subjective state are understandable, e.g., 'I have an allergy,' 'I've been under a lot of pressure to met a deadline,' 'I'm worried about my teenager,' an individual can initiate various coping strategies to deal with the causes. When the origins of the experienced distress are vague or unclear, however, an individual starts searching around for salient cues. If the environment provides a plausible cause in the form of a pungent odor or dense, stuffy air, then an individual can conclude, rightly or wrongly, that the poor quality of the environment is responsible for his physical and psychological discomfort. Notice that this process can occur independently of any specific toxic effects the environment might have on the individual and irrespective of the 'real' cause of the autonomic arousal. All that is required is that an individual experience autonomic arousal in response to a subtle or unidentified stressor or combination of stressors. Cues provided by the environment

in the form of noxious odors, visually detectable particulates or dust, or humid, stuffy air, may suggest to an individual that his discomfort is a toxic response to an airborne pollutant. That environment then becomes a source of threat to the individual, which in turn may generate more autonomic arousal and anxiety.

REFERENCES

1. Melius, J., et al., "Indoor Air Quality -- the NIOSH Experience," Ann Am Conf Gov Ind Hyg 10: 3-7, 1984.

2. Robertson, G., "Source, Nature and Symptomology on Indoor Air Pollutants." In: Indoor and Ambient Air Quality. R. Perry and P. Kirk (eds.). London, Selper Ltd., 311-319, 1988.

3. Kirkbride, J., Sick Building Syndrome: Causes and Effects, Health and Welfare Canada, Ottawa, Canada, October 24, 1985.

4. Collett, C. et al., "A Database of Problem Buildings: Learning by Past Mistakes." In: Present and Future of Indoor Air Quality. C. Bieva, et al. (eds.). Amsterdam, Elsevier Science Publishers, 413-419, 1989.

5. Sterling, T. and Sterling, E., "Investigations on the Effect of Regulating Smoking on Levels on Indoor Pollution and on the Perception of Health and Comfort of Office Workers." In: ETS - Environmental Tobacco Smoke: Report from a Workshop on Effects and Exposure Levels. R. Rylander, Y. Peterson and M.C. Snella (eds.). Published simultaneously in European Journal of Respiratory Diseases, Supplement No. 133, Vol. 65, 17-31, 1984.

6. Sterling, T., et al., "Environmental Tobacco Smoke and Indoor Air Quality in Modern Office Work Environments," Journal of Occupational Medicine 29(1): 57-62, 1987.

7. Kirkbride, J., et al., "Health and Welfare Canada's Experience in Indoor Air Quality Investigation." In: Indoor Air '90: Proceedings of the Fifth International Conference on Indoor Air Quality and Climate. Toronto, Canada, Volume 5, 99-106, July 29-August 3, 1990.

8. U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, Division of Standards Development and Technology Transfer, "The NIOSH Approach to Conducting Indoor Air Quality Investigations in Office Buildings," Indoor Air Quality Selected References, September, 1989.

9. Turner, S. and Binnie, P., "An Indoor Air Quality Survey of Twenty-Six Swiss Office Buildings." In: Indoor Air '90: Proceedings of the Fifth International Conference on Indoor Air Quality and Climate. Toronto, Canada, Volume 4, 27-32, July 29-August 3, 1990.

10. Pimm, P., et al., "Physiological Effects of Acute Passive Exposure to Cigarette Smoke," Arch Environ Health 33(4): 201-213, 1978.

11. Winneke, G., et al., "Psychophysiological Response to Environmental Tobacco Smoke in an Experimental Social Setting." In: Indoor Air Quality. H. Kasuga (ed.). Berlin, Heidelberg, Springer-Verlag, 173-183, 1990.

12. Sterling, T. and Kleven, S., "The Epidemiology of 'Sick Public Buildings'." In: Indoor Air Quality: Symposium. Buenos Aires, National Academy of Sciences of Buenos Aires, 79-96, 1989.

13. Hedge, A., et al., "Indoor Air Quality and Health in Two Office Buildings with Different Ventilation Systems," Environmental International 15: 115-128, 1989.

14. Hawkins, L. and Wang, T., "The Office Environment and the Sick-Building Syndrome," Healthy Buildings 365-371, 1991.

15. Hedge, A., et al., "Breathing-Zone Filtration Effects on Indoor Air Quality and Sick Building Syndrome Complaints," Healthy Buildings 351-357, 1991.

16. Sterling, T.D., et al., "Building Illness in the White-Collar Workplace," International Journal of Health Services 3: 277-287, 1983.

17. Helsing, K.J., et al., "Cure of a Sick Building: A Case Study," Environmental International 15: 107-114, 1989.

18. Hanssen, S. and Mathisen, H., "Sick Building -- A Ventilation Problem?," Indoor Air '87, Proceedings of the 4th International Conference on Indoor Air Quality and Climate, Berlin (West), 17-21 August 1987, Institute of Water, Soil and Air Hygiene 3: 357-361.

19. Beller, M. and Middaugh, J., "Results of an Indoor Air Pollution Investigation," Alaska Medicine 31(4): 148-155, 1989.

20. Norback, D., et al., "Overfrequencies of Skin and Airway Symptoms Among Personnel In Sick Buildings and Polluted Industrial Environments in Relation to Exposure," Indoor Air '87, Proceedings of the 4th International Conference on Indoor Air Quality and Climate, Berlin (West), 17-21 August 1987, Institute of Water, Soil and Air Hygiene 2: 515-519.

21. Norback, D., et al., "The Prevalence of Symptoms Associated with Sick Buildings and Polluted Industrial Environments as Compared to Unexposed Reference Groups Without Expressed

Dissatisfaction," Environment International 15: 85-94, 1989.

22. Norback, D. and Torgen, M., "A Longitudinal Study Relating Carpeting with Sick Building Syndrome," Environmental International 15: 129-135, 1989.

23. Woods, J., "Cost Avoidance and Productivity in Owning and Operating Buildings," Occupational Medicine 4: 753-771, 1989.

24. Kim, I., "Sick Building Syndrome: M.E.'s Seek a Cure," Mech Engineering 112: 53-54, November, 1990.

25. Burge, S., et al., "Sick Building Syndrome: A Study of 4373 Office Workers," Ann Occup Hyg 31: 493-504, 1987.

26. Skov, P., et al., "Influence of Personal Characteristics, Job-Related Factors and Psychosocial Factors on the Sick Building Syndrome," Scand J Environ Health 15: 286-295, 1989.

27. Haghighat, F., et al., "Relationship Between Occupant Discomfort as Perceived and as Measured Objectively," Indoor Environ 1: 112-118, 1992.

28. Colligan, M., "The Psychological Effects of Indoor Air Pollution," Bulletin of the New York Academy of Medicine 57: 1014-1026, 1981.

PHILIP MORRIS RESPONSE TO OSHA RFI

Question 3a

Appendix: Press Reports of "Sick Building Syndrome"

The attached Appendix volumes contain press reports of specific incidents of "sick building syndrome." Complaints from occupants in these buildings have been traced to a number of sources, including: high levels of volatile organic compounds, bacteria, mold, spores, plumbing, roof and air conditioning leaks, high heat, excessive humidity and general unsanitary conditions. Consistent with NIOSH data, few, if any, complaints were related to tobacco smoke. In fact, smoking is prohibited in many of these buildings.

Occupants reported symptoms and complaints ranging from allergies and malaise to pneumonia and death. In some cases, the cause was never actually identified, yet building occupants continue to experience symptoms. Some workers have been forced to change employment or were unable to continue working at all.

The attached reports represent but a small sample of affected buildings in the U.S. Building types include public and private office buildings, schools, hotels, hospitals, courthouses, child care centers, retirement homes and even a homeless shelter.

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For ease of reference, a state-by-state listing of the buildings identified in these illustrative, published reports is attached.

BUILDINGS THAT ARE REPORTEDLY SICK

Volume I

State	City	Building
AK	Anchorage	State Office Building
AK		Indoor skating rink
AZ	Tucson	Amelia Maldonado Elementary School
CA	Anaheim	Pacific Volt
CA	Beverly Hills	The Beverly Plaza Hotel
CA	Burbank	Lockheed Corp.
CA	Chino	California Institution for Men
CA	Duarte	City of Hope Medical Center
CA	El Segundo	El Segundo Airport Towers, Building C
CA	Goleta	Raytheon Co.
CA	Hayward	Helen Turner Child Care Center
CA	Jackson	Amador County Courthouse, District Attorneys Office
CA	Los Angeles	UCLA Medical Center
CA	Los Angeles	Westwood Horizons residential facility
CA	Oakland	Merritt-Peralta Medical Center
CA	Oakland	Alameda County Courthouse
CA	Poway	Midland Elementary School
CA	Richmond	Social Security Administration
CA	Richmond	Richmond Health Center
CA	Sacramento	Bateson Building (State Office Building)

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State	City	Building
CA	Sacramento	Twin Towers -- State Office Buildings CA Depts. of Health Services & Social Services
CA	San Diego	San Diego County Courthouse
CA	San Diego	Income Property Group Office Park Building
CA	San Francisco	Bancroft-Whitney Co.
CA	Stanford	Stanford University Medical Center
CA	Sunnyvale	Lockheed Missile and Space Co.
CA	Westwood	Wadsworth Veterans Administration Medical Center
CA		Lakeside Village Condominiums
CO		Susquehanna Building
CO	Castle Rock	Douglas County School District Administrative Offices
CO	Denver	Federal Building
CO	Lamar	Best Western Cow Palace
CO	Rocky Flats	Rocky Flats Nuclear Weapons Plant (U.S. Department of Energy)
CO	Westminster	City Pool
CT	Bristol	Police - Court Complex
CT	Hartford	Bulkeley High School
CT	Hartford	One Myrtle Street
CT	Hartford	U.S. Post Office
CT	New Britain	New Britain High School
CT	New Haven	Hospital of St. Raphael

State	City	Building
CT	New Haven	Yale Medical School, Dept. of Epidemiology and Public Health
CT	Stamford	West Main Street Shelter
CT		Connecticut insurance company
		Volume II
DC		EEOC Headquarters
DC		U.S. Dept. of Interior
DC		U.S. Information Agency
DC		General Services Administration Building
DC		White House
DC		SSA Payment Center
DC		Parklawn Building, U.S. Dept. of HHS
DC		Madison Building, Library of Congress
DC		EPA Headquarters Waterside Mall
DC		U.S. Housing and Urban Development Building
DC		Frances Perkins Building, U.S. Labor Dept.
DC		Hubert Humphrey Office Building
DC		National Museum of American History, Smithsonian Institution
FL		South Florida Savings & Loan
FL	Bartow	Polk County Courthouse

State	City	Building
FL	Deerfield Beach	House of Insurance
FL	Ft. Lauderdale	Broward County Hospital
FL	Ft. Lauderdale	South Florida Savings Bank
FL	Ft. Lauderdale	Broward County School
FL	Ft. Lauderdale	Broward County Library
FL	Ft. Lauderdale	Paragon Building
FL	Ft. Lauderdale	Broward County Airport Control Center
FL	Gainesville	Veterinary School, Univ. of Florida
FL	Kissimmee	The Hyatt - Orlando Hotel
FL	Lauderdale Lakes	Lauderdale Lakes City Hall
FL	Lauderhill	Lauderhill Mall, State of Florida Offices
FL	Oakland Park	Northridge Medical Plaza, V.A. Clinic
FL	Pensacola	Escambia County Dept. of Public Health
FL	University Station	John and Grace Allen Administration Building University of South Florida
FL	West Palm Beach	City Hall
FL	West Palm Beach	Palm Beach County Governmental Center
FL	Ybor City	Lozano Building Hillsborough County Environmental Protection Commi-ssion
GA	Atlanta	101 Marietta Tower Bldg.
IL	Chicago	Wrigley Building

State	City	Building
IL	Chicago	Social Security Administration Bldg.
IN	Evansville	St. Mary's Medical Center
IN	Indianapolis	Methodist Hospital
IN	Russiaville	West Elementary School
IN		Indiana office complex
IA	Iowa City	University of Iowa Hospitals
IA	Ottumwa	Wapello County and Iowa Depts. of Human Services Building
KS	Belleville	Agricultural Stabilization and Conservation Services; Soil Conservation Service USDA Agencies
KS	Wichita	Pioneer TeleTechnologies
KY	Bowling Green	Richardsville Elementary School
LA	Baton Rouge	Pleasant Hall, Louisiana State University
LA	Bogalusa	Winn-Dixie Grocery Store
ME		Maine Department of Transportation
MD	Bethesda	Bethesda Naval Hospital
MD	College Park	University of Maryland
MD	Hagerstown	Western Maryland Center State Hospital
MD	Rockville	U.S. Public Health Service Building
MD	Waldorf	Thomas Stone High School
MD	Woodlawn	U.S. Health Care Financing Administration, Meadows East Building

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State	City	Building
MD	Woodlawn	City Office Building
		Volume III
MA	Andover	West Elementary School
MA	Boston	Metropolitan State Hospital
MA	Boston	Cambridge Rindge & Latin High School
MA	Norwood	Polaroid
MI	Detroit	Madison Center (Court Building)
MI	Detroit	Airport Hilton Inn
MI	Pontiac	Oakland County Complex
MO	Centralia	Temple Industries Inc.
MO	Jefferson City	Truman State Office Building
MT	E. Helena	Tri-Valley Credit Union Building
NE	Lincoln	Federal Building
NV	Carson City	State Capitol Building
NV	Lake Tahoe	Harvey's Wagon Wheel
NH	Concord	Beaverbrook Elementary School
NH	Nashua	New Searles Elementary School
NJ	Atlantic City	Bally's Park Place Hotel and Casino
NJ	Flemington	Hunterdon Central Regional High School
NJ	Mahwah	Municipal Offices
NJ	Mahwah	Ramapo College

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State	City	Building
NJ	Red Bank	Bell Communications Research Building
NM	Albuquerque	Social Security Administration
NY	Albany	Building 8, State Office Campus Department of Taxation and Finance
NY	Brooklyn	Downstate Medical Center
NY	Hudson County	Whitney Young Elementary School
NY	NYC	General Services Administration
NY	NYC	New York Times Company
NY	NYC	Brandeis High School
NY	Rochester	Eastman Kodak
NY	Rochester	Rochester General Hospital
NY	Schenectady	Wappingers Falls High School
NY	Wappingers Falls	Van Wyck Junior High School
OH	Cincinnati	Alms & Doepke Building
OH	Cincinnati	Goodall Building
OH	Cincinnati	Office Building
OH	Columbus	Northland Terrace Nursing and Rehabilitation Center
OH	Dayton	Wright Patterson Air Force Base
OK	Muskogee	CH Haskell Building, Oklahoma State University
PA	Harrisburg	Sporting Hill Elementary School
PA	Philadelphia	Social Security Administration
RI	Providence	Rhode Island Hospital

State	City	Building
TN	Nashville	Tennessee State Museum
TN	Nashville	Metro Courthouse
TN	Nashville	Nashville Federal Building
TN	Nashville	Artista Records Nashville Headquarters
TX	Austin	Texas Dept. of Housing and Community Affairs
TX	Austin	Austin Hospital
TX	Austin	Texas Board of Pardons and Paroles
TX	Dallas	Ferrell Center, Baylor Univ.
TX	Dallas	Polish Power, Inc.
TX	Houston	Johnson Space Center - NASA
TX	Houston	Judwin Properties Apartment Complexes
TX	Wimberly	Elementary School
UT		VA Hospital
UT	Moab	State Social Services Office Building
UT	Ogden	IRS Service Center
VT	Burlington	University of Vermont College of Medicine
VA	Blacksburg	Cowgill Hall-Virginia Tech
VA	McLean	Franklin Sherman Elementary School
VA	Richmond	Montrose Elementary School
VA	Richmond	Office Building
VA	Arlington	USA Today Headquarters

State	City	Building
VA	Springfield	West Springfield High School
VA	Suffolk	Human Resources Building
WA	Kettle Falls	Kettle Falls High School
WA	Monroe	Monroe Reformatory, Washington Dept. of Corrections
WA	Seattle	Harbourview Medical Center
WA	Seattle	John Hay Elementary School
WA	Seattle	Snohomish County Courthouse
WA	Spokane	City Public Safety Building
WA	Tacoma	Highrise office building
WV	Welch	Mount View High School
WI	Eau Claire	Holiday Inn
WI	Green Bay	Brown County Mental Health Center
WI	Milwaukee	West Milwaukee School District
WI	Milwaukee	809 Building
WI	Port Washington	Ozaukee County Courthouse
WI	Sheboygan	Plastics plant
WI	Traux	Madison Area Technical College

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PHILIP MORRIS RESPONSE TO OSHA RFI

Question 5: "In cases where IAQ investigations have identified a bioaerosol as the etiologic cause of a building-related illness:

(a) Did complaints occur within a specific length of time?

(b) Were there similarities in symptoms among affected individuals which suggested exposure to a specific agent, e.g., _Legionella pneumophila_? Was the etiological agent identified?

(c) What laboratory tests were performed to confirm that a specific bioaerosol was responsible for health complaints?

(d) How was the problem resolved?"

Summary:

A review of some published case studies of health effects related to biological contamination of the indoor environment provides some insight into the process of identifying specific biological agents related to those health effects. Outbreaks of various diseases and illnesses have been correlated with improper maintenance or operation of ventilation systems, suggesting that proper maintenance and operation could reduce the incidence of bioaerosol-related illness. Specifically, _Legionella_, the causative agent of both Legionnaires' disease and Pontiac fever, has been detected in cooling towers and water systems. Microbial contamination of humidifiers and of previously flooded areas has been associated with humidifier fever and hypersensitivity pneumonitis.

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Question 5: "In cases where IAQ investigations have identified a bioaerosol as the etiologic cause of a building-related illness:

(a) Did complaints occur within a specific length of time?

(b) Were there similarities in symptoms among affected individuals which suggested exposure to a specific agent, e.g., Legionella pneumophila? Was the etiological agent identified?

(c) What laboratory tests were performed to confirm that a specific bioaerosol was responsible for health complaints?

(d) How was the problem resolved?"

Response:

A review of some published case studies of health effects related to biological contamination of the indoor environment provides some insight into the process of identifying specific biological agents related to those health effects.

Legionnaires' Disease

The outbreak which gave Legionnaire's disease its name occurred during an American Legion Convention at the Bellevue Stratford Hotel in Philadelphia, PA, in 1976.[1] A total of 221 cases were identified; there were 34 deaths. After a six-month investigative process, the bacterium Legionella pneumophila was

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identified as the causative agent from samples of lung tissue.[2] The outbreak was linked to airborne dissemination of bacteria via the hotel's ventilation system.[1]

A 1978 outbreak of 15 cases of L. pneumophila pneumonia at Baptist Memorial Hospital in Memphis, TN, is also often discussed in the literature.[3,4,5] This epidemic coincided with the use of an auxiliary cooling unit that had not been used for two years. Legionella had proliferated in the tower; when it was operated, Legionella-containing aerosol drifted into and was dispersed by the hospital ventilation system.

O'Mahony, et al., report on their investigation of an English police headquarters building where six cases of Legionnaires' disease were reported.[6] They used a case-control design to find the main area of the building associated with the infection (the cases worked in a communications wing and the controls elsewhere in the building). Microbiological investigations of cooling system water and sludge implicated cooling tower drift and exhaust. Interestingly, sick building syndrome symptoms (influenza-like illnesses and other minor complaints) were also associated with working in the communications wing. Remediation procedures included draining and disinfecting the cooling tower, resiting the ventilation system air intake away from the cooling

tower exhaust, and eventually switching to an air-cooled ventilation system.

In September 1991, 13 cases (one fatal) of Legionnaires' disease were diagnosed among 1,200 employees at a Social Security Administration building in Richmond, CA.[7] Investigators reportedly found Legionella in a basement sink, water fountains, elevator shafts and two of the building's five cooling towers. One case of Legionnaires' disease was also diagnosed in 1991 at the IRS Service Center in Ogden, UT.[7]

Pontiac Fever

In 1968, during a one-week period, 95 of 100 employees contracted a flu-like illness at an Oakland County Health Department building in Pontiac, MI.[3,4,5,8] There was an association between development of the illness and presence in the building when the air-conditioning system was operating. Exhaust from an evaporative condenser was found to be located near the intake of another ventilation system. At the time of the outbreak, the etiologic agent could not be identified, but was eventually traced to Legionella, when that organism was identified following the 1976 Legionnaires' disease outbreak in Philadelphia. This outbreak and several outbreaks of Legionnaires' disease have been traced to airborne transmission of bacteria.[9]

Hypersensitivity Pneumonitis and Humidifier Fever

Finnegan and Pickering correlate the development of hypersensitivity pneumonitis (HP) with the presence of contaminated humidifiers.[5] In one case, four of 27 office workers developed either progressive dyspnea or the symptoms of HP (intermittent chills, fever and shortness of breath while at work). With regard to humidifier fever, in one workplace, 12 of 17 workers showed systemic or respiratory symptoms after exposure to a contaminated humidifier. The symptoms stopped when their exposure to humidifiers stopped and re-appeared when exposure began again; therefore, this led to the conclusion that the humidifier was the source of the causative agent.

In a 1988 review, Morey and Feeley report on several case studies.[10] In one, a large office building had repeated floods and other "water disasters." More than 25% of the occupants exhibited symptoms of humidifier fever. An amoeba (Acanthamoeba polyphaga) and a bacterium (Thermoactinomyces) were isolated; these have been associated with hypersensitivity lung disease elsewhere. Nevertheless, because of sampling difficulties, the etiologic agent in this case could not be conclusively identified. In another case, one occupant of an office was being treated for a respiratory infection caused by Pseudomonas aeruginosa. This bacterium was

not found in a sample taken after the office portable humidifier had been turned off for at least twelve hours. However, a "dramatic" elevation in indoor bacteria followed within a few minutes after the humidifier was turned on, suggesting that the source of the infection-causing bacteria was the humidifier.

Laboratory tests

Tests may be performed on samples to isolate specific agents.[11] Culture media may be used to isolate individual types of organisms, by providing certain nutritional requirements specific to the species. Collected aerosols may be examined using antigen analysis when a causal organism is strongly suspected and when specific antibodies are available, e.g., in the case of HP or humidifier fever. Specific analyses for toxins, e.g., endotoxin are being developed.

The Enzyme Linked Immuno-Sorbent-Assay (ELISA) inhibition test is useful in identifying airborne allergens.[12] Also, the RadioImmunoSorbent Test (RAST), an in vitro method, can be used to assess allergenicity.[13]

REFERENCES

1. American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc., Legionellosis Position Statement/Legionellosis Position Paper, ASHRAE, Atlanta, GA, 1989.

2. Winn, W.C., "Legionnaires Disease: Historical Perspective," Clinical Microbiology Reviews 1: 60-81, 1988.

3. Imperato, P.J., "Legionellosis and the Indoor Environment," Bulletin of the New York Academy of Medicine 57: 922-935, 1981.

4. LaForce, F.M., "Airborne Infections and Modern Building Technology," Environment International 12: 137-146, 1986.

5. Finnegan, M.J. and Pickering, C.A.C., "Review: Building Related Illness," Clinical Allergy 16: 389-405, 1986.

6. O'Mahony, M., Lakhani, A., Stephens, A., Wallace, J.G., Youngs, E.R. and Harper, D., "Legionnaires' Disease and the Sick-Building Syndrome," Epidem Inf 103: 285-292, 1989.

7. Wallace, B., "No Proof Yet of Source of Legionnaire's Disease," The San Francisco Chronicle, A12, September 21, 1991.

   "Bacteria Keeps Building Closed," Los Angeles Times, A27, September 21, 1991.

   "Second Suspected Outbreak of Legionnaire's Disease Demands Massive Government Intervention, Union Chicago," PR Newswire, September 20, 1991.

   Johnson, C., "Legionnaire's Disease Cleanup Moving Slowly, Federal Office May Be Closed Three Weeks," The San Francisco Chronicle, A20, September 20, 1991.

   "Legionnaire's Disease Cases Reach Nine," The Associated Press, September 19, 1991.

   "Legionnaires' Cases at Building Rise to 9," Los Angeles Times, September 19, 1991.

   Johnson, C., "8th New Case of Legionnaire Disease Found," The San Francisco Chronicle, A17, September 19, 1991.

   Goodavage, M., "Workers Fear Return to 'Sick Building,'" USA Today, 3A, September 20, 1991.

8. Bardana, E.J., Montanaro, A. and O'Hollaren, M.T., "Building-Related Illness: A Review of Available Scientific Data," Clinical Reviews in Allergy 6: 61-89, 1988.

9. Blackmon, J.A., Chandler, F.W., Cherry, W.B., England, A.C., Feeley, J.C., Hicklin, M.D., McKinney, R.M. and Wilkinson, H.W., "Legionellosis," American Journal of Pathology 103(3): 427-465, 1981.

10. Morey, P.R. and Feeley, J.C., "Microbiological Aerosols Indoors, in Workplaces, Schools, and Homes," ASTM Standardization News Dec. 1988: 54-58, 1988.

11. Burge, H.A., "Bioaerosols: Prevalence and Health Effects in the Indoor Environment," The Journal of Allergy and Clinical Immunology 86: 687--701, 1990.

12. Elixmann, J.H., Linskens, H.F., Schata, M. and Jorde, W., "Can Airborne Fungal Allergens Pass Through an Air-Conditioning System?" Environment International 15: 193-196, 1989.

13. van Bronswijk, J.E.M.H., Rijckaert, G. and van de Lustgraaf, B., "Indoor Fungi, Distribution and Allergenicity," Acta Bot. Neerl. 35: 329-345, 1986.

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PHILIP MORRIS RESPONSE TO OSHA RFI

Question 6: "IAQ investigations conducted by NIOSH indicate that some type of biological contaminant was involved in five percent of the cases.

(a) Are there other data available which indicate the prevalence of biological contaminants as the cause of adverse health effects?

(b) Are data available which indicate the likelihood that health complaints are related to a specific bioaerosol contaminant? If so, please indicate the source of such data."

Summary:

Publicly available statistics indicate that a substantial portion (around 40%) of buildings may have biological contamination. Some 8-10% of pneumonia cases in the United States may be due to Legionella, a bacterium often found in water-containing components of ventilation systems. Large numbers of individuals may experience symptoms of asthma or sick building syndrome, or may become ill with colds or flu; such conditions affect their ability to work and result in health care and other costs. For instance, some researchers estimate that overall costs related to respiratory illnesses in the United States may reach into the billions of dollars. Bioaerosol contamination of ventilation systems and subsequent illness among exposed persons is thus a major concern.

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PHILIP MORRIS RESPONSE TO OSHA RFI

Question 6: "IAQ investigations conducted by NIOSH indicate that some type of biological contaminant was involved in five percent of the cases.

(a) Are there other data available which indicate the prevalence of biological contaminants as the cause of adverse health effects?

(b) Are data available which indicate the likelihood that health complaints are related to a specific bioaerosol contaminant? If so, please indicate the source of such data."

<u>Response</u>:

<u>Prevalence of Building Complaints Referable to Bioaerosols</u>

A recent article in <u>Healthy Buildings International</u> (HBI) magazine stated that in Honeywell's Indoor Air Quality Diagnostics group's investigation of 51 commercial office buildings between 1986 and 1990, microbial contamination was considered a factor in 41% of the cases.[1] Similarly, HBI's own investigation of over 700 buildings identified 43% with microbial contamination (34% fungal, 9% bacterial).

The state of California instituted a telephone log to track complaints received by its indoor air quality program.[2] Calls

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to this program suggested that 16% of building occupants with a complaint perceived that biological contamination was a problem.

Bardana, et al., reference other sources of data on building investigations in addition to NIOSH: the Centers for Disease Control has investigated 12 building-associated disease outbreaks between 1978 and 1981, and the State of Oregon Department of Human Resources (Health Division) has investigated 11 since 1984.[3] They also note a number of investigations sponsored by state agencies, including the State Department of Transportation building, Augusta, ME; the Health Service Center, Sandpoint, ID; the Human Services building, Holland, MI; the NBC offices, New York, NY; the Gregory Bateson Building, Sacramento, CA; the Social Services building, San Francisco, CA; and Oakland High School, Oakland, CA.

Prevalence of Diseases/Symptoms Referable to Bioaerosols

Burge and Hodgson report that 8-10% of community-acquired pneumonias are attributable to *Legionella*.[4] Case-fatality rates are approximately 15%, making these bacteria "a major contributor to mortality in the U.S.: an estimated 5,000 to 7,000 deaths per year." In the 85% of non-fatal cases, legionellosis has a "substantial financial burden through time lost from work and additional health care costs." These authors also estimate costs related to episodes of viral respiratory disease in the range of

150 million work days and $59 billion. With regard to allergic asthma, which may be exacerbated by bioaerosols, they estimate that 3% of the U.S. population (9,000,000 persons) has this condition. Of 135,000 hospital admissions yearly, averaging 8 days and costing 5,000,000 work days per year, approximately 15% are due to allergic disease. Burge and Hodgson find the data on HP to be less well-established. Two studies of office workers suggested that workers reported symptoms consistent with HP at rates of 1.2 or 4%. Similarly, humidifier fever complaint rates are reportedly in the area of 2-3%.

Tamblyn and colleagues recently reported that various surveys had reported that the prevalence of nasal problems among workers in mechanically-ventilated office buildings was 22-62% and cough was 18-41%.[5] In their own survey of four buildings for fungal spore content, the prevalence of weekly respiratory and mucosal irritation symptoms among 1,627 persons ranged from 24-47%.

Burge tabulates available statistics on the indoor transmission of respiratory diseases.[6] She estimates that 800,000,000 persons suffer a respiratory illness each year (many of these influenza and the common cold), with an estimated total cost of $24,000,000,000. With the "conservative" estimate that 10% are due to IAQ, a cost of $750,000,000 is attributed to indoor air quality. For Legionnaires' disease, she estimates 20,000

community and 200,000 hospital cases each year, with about 36,000 deaths. 90% would be related to IAQ. Burge also states that at least 35,000 deaths per year can be attributed to airborne infectious disease; related costs are over $1 billion. Influenza results in 400 million days of restricted activity (almost 2 days per person per year). 53-71% of infected adults have lower respiratory symptoms, resulting in lost work time and hospitalization.

REFERENCES

1. Anonymous, "Biological Pollutants in the Indoor Environment," Healthy Buildings International Magazine Sept./Oct. 1991: 10-13, 1991.

2. Macher, J.M., "Inquiries Received by the California Indoor Air Quality Program on Biological Contaminants in Buildings." In: Advances in Aerobiology. Basel, Birkhauser Verlag, 275-278, 1987.

3. Bardana, E.J., Montanaro, A. and O'Hollaren, M.T., "Building-Related Illness: A Review of Available Scientific Data," Clinical Reviews in Allergy 6: 61-89, 1988.

4. Burge, H.A. and Hodgson, M., "Health Risks of Indoor Pollutants," ASHRAE Journal 30: 34-36, 38, 1988.

5. Tamblyn, R.M., Menzies, R.I., Comtois, P., Hanley, J., Tamblyn, R.T., Farant, J.P. and Marcotte, P., "A Comparison of Two Methods of Evaluating the Relationship Between Fungal Spores and Respiratory Symptoms Among Office Workers in Mechanically Ventilated Buildings." In: IAQ 91: Healthy Buildings. Atlanta, American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc., 136-141, 1991.

6. Burge, H.A., "Risks Associated with Indoor Infectious Aerosols," Toxicology and Industrial Health 6: 263-274, 1990.

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PHILIP MORRIS RESPONSE TO OSHA RFI

Question 7: "Persons with underlying health problems or chemical sensitivities often cannot work in industries where physical strength and endurance or exposure to chemicals occur in the normal job experience.

(a) Is there evidence to suggest that these persons are more susceptible to developing health effects due to short-term exposure to PTS, such as eye and respiratory tract irritation?

(b) Is there evidence to suggest that these persons are more susceptible to developing health effects due to long-term exposure to PTS, such as cardiovascular disease and lung cancer?"

Summary:

There is a growing controversy in the medical community over the nature and extent of a physiological condition known as multiple chemical sensitivity (MCS). Conventional physicians and "clinical ecologists" disagree on the origins of the alleged sensitivities. Many conventional physicians appear to believe that the condition may be of a psychological origin. We are unaware of any original data on whether people allegedly afflicted with MCS are at increased risk of long-term health effects associated with exposure to various indoor air constituents. There are reports in the scientific literature, however, that people supposedly afflicted with MCS are more reactive and sensitive to short-term exposures to a wide range of indoor air constituents, outdoor air constituents, food additives and water contaminants. Authors of various studies have reported that adequate ventilation and focusing on indoor air in its entirety will help avoid symptoms of MCS allegedly due to tight building syndrome.

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Question (7): "Persons with underlying health problems or chemical sensitivities often cannot work in industries where physical strength and endurance or exposure to chemicals occur in the normal job experience.

(a) Is there evidence to suggest that these persons are more susceptible to developing health effects due to short-term exposure to PTS, such as eye and respiratory tract irritation?

(b) Is there evidence to suggest that these persons are more susceptible to developing health effects due to long-term exposure to PTS, such as cardiovascular disease and lung cancer?"

<u>Response</u>:

We are unaware of any studies reporting original scientific data on persons allegedly afflicted with multiple chemical sensitivity (MCS). This is due, in part, to the fact that there is not a widely accepted definition of the condition (e.g., as Hileman wrote, "there is a lack of a clear definition of what it [MCS] is").[1] A second author offered the following possible definition of MCS:[2]

> Multiple chemical sensitivities (MCS) is an acquired disorder characterized by recurrent symptoms, referable to multiple organ systems, occurring in response to demonstrable exposure to many chemically unrelated compounds at doses far below those established in the general

population to cause harmful effects. No single widely accepted test of physiologic function can be shown to correlate with symptoms.

Probably the most heated debate, however, centers on whether MCS is a psychological condition, a physiological condition, or both.[1-2] Hileman wrote the following regarding this controversy:[1]

> To many traditional allergists and psychiatrists, the onset of MCS is due almost entirely to psychological factors. Some consider the syndrome to be a variant of somatoform disorder (the conversion of mental experiences into bodily symptoms) or of posttraumatic stress disorder. Others say that the root problem is an irrational belief on the part of the patients that chemicals are making them sick. . . Other physicians -- and these are likely to be clinical ecologists and some traditional physicians who practice occupational medicine -- believe that chemical exposures are causing these people to be ill. They think so because of common threads they see in the clinical histories of these patients.

Terr wrote the following in her critique of clinical ecology theories and practice:[3]

> The concept of multiple chemical hypersensitivities as a disease entity in which the patient experiences numerous symptoms from numerous chemicals and foods caused by a disturbance of the immune systems lacks a scientific foundation. Published reports of such cases are anecdotal and without proper controls. There is no convincing evidence for any immunologic abnormality in these cases.

> Diagnostic methods have been shown to be unreliable. Diagnosis, treatment, and theoretical concepts underlying the purported disease are not consistent with current immunologic knowledge and theory. As defined and presented by its proponents, multiple chemical hypersensitivities constitutes a belief and not a disease.

Brodsky wrote the following regarding the continuing controversy in the medical field:[4]

> The clinical ecology subculture, like earlier medical subcultures, is the product of patient concerns that the medical establishment cannot allay by treatment or by reassurance. For social and behavioral scientists, it represents a 'natural' experiment that can be studied. For those physicians who believe that clinical ecology is without scientific basis generally and/or that its practitioners interpret laboratory results incorrectly, it is a challenge and an irritant. The clinical ecologist-physician feels rejected and the victim of bias and unfair attack. The patient in this subculture feels that finally he has found someone who understands him and is trying to help him, but that he must pay the price of being disapproved or rejected by his former physicians.

In fact, Hileman wrote that "a large part of the medical profession has become so convinced of the ineffectiveness of clinical ecologists' therapies that physicians who suggest to patients ideas or treatments that are uniquely associated with clinical ecology risk being disciplined by their local medical boards."[1] However, MCS has been "legitimized in a number of federal and state

government regulations and proposed bills."[1] The Social Security Administration also recognizes MCS, but the agency requires a case-by-case analysis of this illness before it will allow payment of disability.[1]

What is MCS?

Hileman describes MCS as follows:[1]

> As a group, people allegedly suffering from MCS have a large number and range of symptoms they attribute to chemical exposures. The complaints are physical and mental and involve nearly all the systems of the body. The most common symptoms include respiratory problems; headache; fatigue; flulike symptoms; mental confusion and short-term memory loss; gastrointestinal tract difficulties; cardiovascular irregularities; skin disorders; genitourinary problems; muscle and joint pains; irritability and depression; and eye, ear, nose, and throat problems. Some individuals experience only one symptom, but most have more than one.

She wrote that the "number of chemicals the victims respond to is equally wide." Hileman asserts that "in some of these complex mixtures it is hard to tell what, if anything, the patient is responding to." She stated the following regarding the specific circumstances that patients believe caused them to be afflicted with MCS:

> Many patients can identify specific circumstances that initiated their illness. They say their problems began either after an overwhelming chemical exposure, such as a job-related chemical spill, or after a new, chronic, medium-level exposure, such as moving into a new house with significant emissions of volatile organic compounds from the building materials or beginning to work in a new building, often one with poor ventilation. Thereafter, the symptoms seem to wax and wane with low-level chemical exposures, and when exposures are avoided altogether, the symptoms abate or disappear. In an informal survey of some 6800 persons claiming to be chemically sensitive, conducted by a patient group called the National Foundation for the Chemically Hypersensitive, nearly half of the patients say their illness started with a pesticide exposure.

Hileman reports that, in the literature, people with MCS can be divided into four categories: 1) industrial workers; 2) tight building occupants; 3) contaminated communities; and 4) individuals. Ashford reports that two general types of exposure seem particularly able to initiate hypersensitivity: 1) "A massive, overwhelming exposure, such as a chemical spill, a fire involving synthetic materials, pesticide spraying, or working with chemicals in a confined, unventilated space"; and 2) "Repeated, low-level exposure to a complex array of synthetic organic compounds, as occurs with combustion products (such as diesel exhaust), tight buildings, and soldering."[5] Ashford wrote the following regarding "trigger" exposures in MCS:

At home, troublesome exposures for the chemically sensitive patient include the gas stove, one of the most commonly identified triggers of symptoms in these patients; combustion products from gas- or oil-fired furnaces and space heaters, water heaters, and central air heating systems; sponge rubber bedding, padding, and upholstery; plastics (especially pliable odorous plastics such as shower curtains); insecticides; perfumes; paintings and decorating materials; fireplaces; cleaning agents; disinfectants; deodorizers; mothballs; cedar closets; newsprint and other printed materials; fabrics in clothing, bedding, and window coverings, especially synthetics or coated fabrics; particleboard; gasoline vapors from attached garages; and carpeting and carpet padding. Disinfectant liquids and sprays containing phenolics frequently provoke symptoms in these patients.

Ashford asserted that in the workplace, adequate ventilation is very important.[5] He cited a study that compared health problems in two office buildings, one fully air-conditioned and the other naturally ventilated. The authors reported that "sickness was significantly increased in the air-conditioned building versus the naturally ventilated building." Ashford also discussed the effects of indoor air pollution in the EPA headquarters in Washington, D.C. in 1987 and 1988. He reported that "an estimated 124 of 2,000 employees exposed to volatile off-gassing from the carpet became ill, exhibiting symptoms ranging from eye, nose, and throat irritation and breathing problems to nausea, headache, dizziness, difficulty in thinking, fatigue, and increased susceptibility to many exposures formerly tolerated."

Cullen wrote the following regarding lack of adequate ventilation in office buildings:[2]

> Two other important and recently recognized clinical syndromes in occupational medicine also deserve mention vis à vis MCS: tight building syndrome and mass psychogenic illness. In the tight building syndrome, now occurring in epidemic proportion in modern offices and other work places, low levels of irritants combine with inadequate fresh-air intake and often low humidity to cause irritative symptoms often associated with CNS problems such as fatigue and poor concentration.

Mooser wrote that "while some individuals have developed MCS following tight building syndromes, the overwhelming majority has not."[6] He states that "the incidents are instructive, however, in that they provide evidence of the irritant effects of low-level exposures, as well as the variability of individual reactivity." He also wrote:

> Although the syndrome of multiple chemical sensitivities has received increasing attention in recent years, data concerning its prevalence among the general population is virtually non-existent. Lack of a common definition as well as the variability of symptoms and diagnostic criteria hamper this effort. Little data is available the characterizes the multiple chemical sensitive population, although incidence among females appears to be considerably higher. Further research is needed not only to better characterize the population but, more importantly, to evaluate potential triggering factors.

Levin wrote the following regarding the etiologic considerations of "environmental illness":[7]

> The potential causal agent must be one that can realistically initiate an illness. If a patient whose history, physical findings, and laboratory results are consistent with a diagnosis of environmental illness described himself as completely well until he was exposed to the second-hand smoke of a single cigarette, another potential etiologic factor would be sought. If this patient described the onset of illness after a single exposure to an intensely toxic gas that is associated with severe acute symptoms, the toxic gas would be a candidate as the causal factor, since this is an etiologic agent that can reasonably cause disease.

He also wrote that "adequate ventilation of workplaces, utilization of appropriate protective clothing and respirators for workers, coupled with appropriate waste disposal techniques, will avoid contamination of workers and others with toxic chemicals and the spread of problems."

<u>Summary</u>

In summary, there are no original data on whether people allegedly afflicted with MCS are at increased risk of long-term health effects associated with exposure to various indoor air constituents. There are reports in the literature, however, that people reportedly afflicted with MCS are more reactive and

REFERENCES

1. Hileman, B., "Multiple Chemical Sensitivity," C & EN July 22: 26-42, 1991.

2. Cullen, M.R., "The Worker with Multiple Chemical Sensitivies: An Overview," Occupational Medicine: State of the Art Reviews 2(4): 655-661, 1987.

3. Terr, A., "'Multiple Chemical Sensitivities': Immunologic Critique of Clinical Ecology Theories and Practice," Occupational Medicine: State of the Art Reviews 2(4): 683-694, 1987.

4. Brodsky, C., "Multiple Chemical Sensitivies, and Other 'Environmental Illness': A Psychiatrist's View," Occupational Medicine: State of the Art Reviews 2(4): 695-704, 1987.

5. Ashford, N.A. and Miller, C.S., "Chemical Exposures: Low Levels and High Stakes. Part I. Defining Chemical Sensitivity: Chapter 3. Origins of Multiple Chemical Sensitivity and Effects on Health," Chemical Exposures 0(0): 59-84, 1991.

6. Mooser, S.B., "The Epidemiology of Multiple Chemical Sensitivities (MCS)," Occupational Medicine: State of the Art Reviews 2(4): 663-668, 1987.

7. Levin, A.S. and Byers, V.S., "Environmental Illness: A Disorder of Immune Regulation," Occupational Medicine: State of the Art Reviews 2(4): 669-681, 1987.

sensitive to short-term exposures to a wide range of indoor air constituents, outdoor air constituents, food additives, and water contaminants. Authors of various studies have reported that adequate ventilation and focusing on indoor air in its entirety will help avoid future cases of MCS allegedly due to tight building syndrome.

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PHILIP MORRIS RESPONSE TO OSHA RFI

Question 8: "Some people may develop an increased sensitivity to chemical pollutants, such as found in PTS, during pregnancy or treatment with certain medications (Calabrese 1978). What additional studies pertain to this sensitivity?"

Summary:

Calabrese (cited in OSHA RFI, reference #2) suggests that pregnant women and people on certain medications may develop an increased sensitivity to a wide-range of chemical substances. We are unaware of any other scientific studies regarding these alleged sensitivities. Contrary to the question above, Calabrese does not appear to implicate PTS per se as a specific agent related to these sensitivities. The author states that cadmium, lead, manganese, carbon monoxide, organophosphate insecticides, beryllium, fluoride, nitrogen dioxide, sulfur dioxide and ozone may potentially be associated with sensitivity reactions in some individuals. Although a few of these chemicals are reportedly found in tobacco smoke, there are numerous potential sources of these chemicals in the indoor environment.

PHILIP MORRIS RESPONSE TO OSHA RFI

Question 8: "Some people may develop an increased sensitivity to chemical pollutants, such as found in PTS, during pregnancy or treatment with certain medications (Calabrese 1978). What additional studies pertain to this sensitivity?"

Response:

We are unaware of any other published studies that examine the sensitivities discussed by Calabrese in 1978. Furthermore, Calabrese does not appear to implicate PTS per se as a specific agent related to the sensitivities discussed in his paper. However, Calabrese does suggest that certain substances, including several that are sometimes found in the indoor environment, may be associated with sensitivity reactions among certain individuals.

Calabrese discusses developmental factors, genetic factors, dietary deficiencies, diseases, and behavioral factors that may predispose certain individuals to the possible toxic effects of environmental-occupational pollutants. The author provided charts of suspected pollutant agents and their possible health effects. PTS per se was not listed on these charts. Although several of the chemicals listed are reportedly found in

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tobacco smoke, there are numerous potential sources of these chemicals in the indoor environment.

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PHILIP MORRIS RESPONSE TO OSHA RFI

Question 11: "To your knowledge, have PTS exposures been associated with specific adverse health effects in humans?"

Response:

See Response to OSHA RFI Question 2a(iii) for a full discussion and critique of the literature on PTS exposures and reported associations between spousal (and parental) smoking and chronic/acute disease endpoints.

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PHILIP MORRIS RESPONSE TO OSHA RFI

Question 12: "To your knowledge, have PTS exposures been associated with specific adverse health endpoints in experimental animals?"

Summary:

Animal inhalation experiments using fresh sidestream smoke or constituents of sidestream smoke do not support the claim that PTS is a pulmonary carcinogen. Sidestream smoke is not equivalent to PTS, nevertheless, the two inhalation experiments using sidestream smoke that have been published reported no meaningful differences in the lung tissue of animals exposed to sidestream smoke and those not exposed to smoke. In addition, recent reviews of the literature on suspected pulmonary carcinogens have concluded that none of the individual constituents in sidestream smoke which are classified as potentially carcinogenic have been found to induce pulmonary cancer via inhalation in experimental animals.

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PHILIP MORRIS RESPONSE TO OSHA RFI

Question 12: "To your knowledge, have PTS exposures been associated with specific adverse health endpoints in experimental animals?"

<u>Response</u>:

Animal <u>inhalation</u> experiments using fresh sidestream smoke or constituents of sidestream smoke do not support the claim that PTS is a pulmonary carcinogen. Sidestream smoke is <u>not</u> equivalent to PTS, nevertheless, the two inhalation experiments using sidestream smoke that have been published reported no meaningful differences in the lung tissue of animals exposed to sidestream smoke and those not exposed to smoke. In one of the studies, German scientists exposed rats and hamsters to very high levels of sidestream smoke for a 90-day period; they reported no significant physiological effects on the tissues of the animals.[1] In the second study, researchers from the American Health Foundation exposed hamsters to sidestream and mainstream smoke for 18 months; they observed no significant increase in lung tumors among the animals exposed to sidestream smoke.[2,3,4]

In addition, recent reviews of the literature on suspected pulmonary carcinogens have concluded that <u>none</u> of the individual constituents in sidestream smoke which are classified as potentially

carcinogenic have been found to induce pulmonary cancer via inhalation in experimental animals.[5,6]

Positive results reported for skin painting, intra-tracheal implantation or subcutaneous application in animals are of questionable relevance to the mode of exposure (inhalation) for PTS.

<u>REFERENCES</u>

1. Adlkofer, F., et al., "Exposure of Hamsters and Rats to Sidestream Smoke of Cigarettes: Preliminary Results of a 90-Day-Inhalation Study." In: <u>Indoor and Ambient Air Quality</u>. R. Perry and P. Kirk (eds.). London, Selper Ltd., 252-258, 1988.

2. Haley, N., et al., "Uptake of Sidestream Smoke by Syrian Golden Hamsters," <u>Toxicol Letters</u> 35: 83-88, 1987.

3. Haley, N., "Sidestream Smoke Uptake By Syrian Golden Hamsters in an Inhalation Bioassay." In: <u>Indoor Air '87: Proceedings of the 4th International Conference on Indoor Air Quality and Climate</u>. Berlin, Volume 2, 68-73, August 17-21, 1987.

4. Hoffmann, D., American Health Foundation, "Experimental Tobacco Carcinogenesis: 2 P01 CA29580-07, Subproject 6: Sidestream Smoke Carcinogenesis," Application for Continuation Grant to U.S. Department of Health and Human Services, Public Health Service, April 29, 1988.

5. Aviado, D., "Suspected Pulmonary Carcinogens in Environmental Tobacco Smoke," <u>Environ Tech Letters</u> 9: 539-544, 1988.

6. Aviado, D., "Health Effects of 50 Selected Constituents of Environmental Tobacco Smoke." In: <u>Indoor Air Quality</u>. H. Kasuga (ed.). Berlin, Heidelberg, Springer-Verlag, 383-389, 1990.

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PHILIP MORRIS RESPONSE TO OSHA RFI

Question 13(a): "Have these factors been considered in instances where IAQ investigations have failed to identify a specific contaminant source?

(b) If yes, was remedial action taken to improve these conditions? Please explain what that action was.

(c) Did health complaints decline?"

<u>Summary</u>:

Sick building investigation databases indicate the important role of so-called physical stressors such as temperature, lighting, noise, and ergonomics in perceived worker satisfaction with the working environment. Job satisfaction, security, and type of work also are important factors in perceived worker comfort. Tobacco smoke, because it is visible and easily identifiable, is often identified as a "stressor" in the work environment. However, investigations reveal that reported complaints about tobacco smoke may be misdirected and the result of less obvious conditions such as lack of ventilation (stuffy air), high temperatures, inadequate lighting, overcrowding and uncomfortable working conditions. Increased outdoor ventilation, as well as adequate temperature and humidity control, have resulted in a decrease in worker complaints about IAQ.

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PHILIP MORRIS RESPONSE TO OSHA RFI

Question 13(a): "Have these factors been considered in instances where IAQ investigations have failed to identify a specific contaminant source?

(b) If yes, was remedial action taken to improve these conditions? Please explain what that action was.

(c) Did health complaints decline?"

Response:

Sick building investigation databases indicate the important role of so-called physical stressors such as temperature, lighting, noise, and ergonomics in perceived worker satisfaction with the working environment.[1-8] Job satisfaction, security, and type of work also are important factors in perceived worker comfort.[9-13] Tobacco smoke, because it is visible and easily identifiable, is often identified as a "stressor" in the work environment. However, investigations reveal that reported adverse response to tobacco smoke may be misdirected and a result of less obvious conditions such as lack of ventilation (stuffy air), high temperatures, inadequate lighting, overcrowding and uncomfortable working conditions.[1-13] Increased outdoor ventilation, as well as adequate temperature and humidity control, have resulted in a decrease in worker complaints about IAQ.

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REFERENCES*

1. Melius, J., et al., "Indoor Air Quality -- the NIOSH Experience," Ann Am Conf Gov Ind Hyg 10: 3-7, 1984.

2. Robertson, G., "Source, Nature and Symptomology on Indoor Air Pollutants." In: Indoor and Ambient Air Quality. R. Perry and P. Kirk (eds.). London, Selper Ltd., 311-319, 1988.

3. Kirkbride, J., Sick Building Syndrome: Causes and Effects, Health and Welfare Canada, Ottawa, Canada, October 24, 1985.

4. Collett, C., et al., "A Database of Problem Buildings: Learning by Past Mistakes." In: Present and Future of Indoor Air Quality. C. Bieva, et al. (eds.). Amsterdam, Elsevier Science Publishers, 413-419, 1989.

5. Sterling, T. and Sterling, E., "Investigations on the Effect of Regulating Smoking on Levels on Indoor Pollution and on the Perception of Health and Comfort of Office Workers." In: ETS - Environmental Tobacco Smoke: Report from a Workshop on Effects and Exposure Levels. R. Rylander, Y. Peterson and M.C. Snella (eds.). Published simultaneously in European Journal of Respiratory Diseases, Supplement No. 133, Vol. 65, 17-31, 1984.

6. Sterling, T., et al., "Environmental Tobacco Smoke and Indoor Air Quality in Modern Office Work Environments," Journal of Occupational Medicine 29(1): 57-62, 1987.

7. Kirkbride, J., et al., "Health and Welfare Canada's Experience in Indoor Air Quality Investigation." In: Indoor Air '90: Proceedings of the Fifth International Conference on Indoor Air Quality and Climate. Toronto, Canada, Volume 5, 99-106, July 29-August 3, 1990.

8. U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, Division of Standards Development and Technology Transfer, "The NIOSH Approach to Conducting Indoor Air Quality Investigations in Office Buildings," Indoor Air Quality Selected References, September, 1989.

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* Copies of listed references are supplied in the Philip Morris Response to RFI Question No. 3.

9. Pimm, P., et al., "Physiological Effects of Acute Passive Exposure to Cigarette Smoke," Arch Environ Health 33(4): 201-213, 1978.

10. Winneke, G., et al., "Psychophysiological Response to Environmental Tobacco Smoke in an Experimental Social Setting." In: Indoor Air Quality. H. Kasuga (ed.). Berlin, Heidelberg, Springer-Verlag, 173-183, 1990.

11. Sterling, T.D., et al., "Building Illness in the White-Collar Workplace," International Journal of Health Services 3: 277-287, 1983.

12. Skov, P., et al., "Influence of Personal Characteristics, Job-Related Factors and Psychosocial Factors on the Sick Building Syndrome," Scand J Environ Health 15: 286-295, 1989.

13. Colligan, M., "The Psychological Effects of Indoor Air Pollution," Bulletin of the New York Academy of Medicine 57: 1014-1026, 1981.

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PHILIP MORRIS RESPONSE TO OSHA RFI

Question 14: "If your company keeps records of employee IAQ complaints, can you summarize your experience, emphasizing your efforts to localize the problem, identify the contaminants, determine the adverse health effects, and action taken?"

Summary:

Philip Morris retains information on specific IAQ investigations that have been conducted, although few IAQ complaints have been recorded. To localize potential problems, an investigation usually begins in an area where employees have complained of symptoms (e.g., eye, nose or throat irritation, dizziness, headache or nausea). A preliminary walk-through is conducted, to determine if any ongoing processes could be contributing a contaminant to the work area. This is followed by a visual survey of the HVAC system in that area, to determine the supply and exhaust layout and the quantities of air supplied to the work area. The HVAC system is checked to ensure that it is operating properly and to determine the percentage of fresh make-up air. Air filters and condensate collection pans are also examined. When environmental measurements are conducted, substances measured may include oxygen, carbon dioxide, carbon monoxide, formaldehyde and ozone.

PHILIP MORRIS RESPONSE TO OSHA RFI

Question 14: "If your company keeps records of employee IAQ complaints, can you summarize your experience, emphasizing your efforts to localize the problem, identify the contaminants, determine the adverse health effects, and action taken?"

Response:

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